IN THE SUPERIOR COURT OF THE STATE OF CALIFORNIA
IN AND FOR THE COUNTY OF SAN DIEGO
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Coordination Proceeding Special Title (Rule 1550(b))	JCCP No. 4042
In re TOBACCO CASES II	
This document relates to: The People of the State of California, et al. v. Philip Morris, Incorporated, et al., Los Angeles Superior Court Case No. BC 194217;	
The People of the State of California, et al. v. General Cigar Co., et al., San Francisco Superior Court Case No. 996780;	
The People of the State of California, et al. v. Brown & Williamson, et al., San Francisco Superior Court Case No. 996781;	
The People of the State of California, et al. v. Tobacco Exporters, et al., San Francisco Superior Court Case No. 301631.	

VIDEOTAPED EXPERT DEPOSITION OF

KENT E. PINKERTON, Ph.D.

Monday, May 22, 2000

(Volume I - Pages 1 through 185)

REPORTED BY: SHANNON TAYLOR-SCOTT, RPR, CSR 10067
JOB NO. 05-93617

I N D E X

INDEX OF EXAMINATIONS

DEFENDANTS' EXHIBITS MARKED FOR IDENTIFICATION

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and additional Plaintiffs.)
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BE IT REMEMBERED that, pursuant to Notice, and on Monday, May 22, 2000, commencing at 9:37 a.m. thereof, at COMBS & GREENLEY, INC., 2520 Venture Oaks Way, Suite 220, Sacramento, California 95833, before me, SHANNON TAYLOR-SCOTT, a Certified Shorthand Reporter, personally appeared

KENT E. PINKERTON, Ph.D

called as a witness by the Defendant RJ Reynolds Tobacco Company, who, having been first duly sworn, was examined and testified as follows:

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APPEARANCES OF COUNSEL:

FOR THE PLAINTIFFS THE PEOPLE OF THE STATE OF CALIFORNIA AND AMERICAN ENVIRONMENTAL SAFETY INSTITUTE AND THE WITNESS:

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ALSO PRESENT:
ALECIA L. MOORE, RESEARCH ANALYST
VIDEO SOLUTIONS, a LegaLink Company
DAVID JENSEN, VIDEOGRAPHER

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P R O C E E D I N G S

09:37:22 THE VIDEOGRAPHER: Here begins Videotape
09:37:24 Number 1 in the deposition of Kent Pinkerton in the
09:37:26 matter of {The People of the State of California versus
09:37:29 Philip Morris, Incorporated, et al., General Cigar
09:37:33 Company, et al., Brown and Williamson, et al., Tobacco
09:37:39 Exporters, et al., in the Superior Court of the State
09:37:41 of California, for the County of San Diego, JCCP
09:37:44 Number 4042.}
09:37:46 Today's date is May 22nd, 2000. The time on
09:37:50 the video monitor is 9:37. The video operator today is
09:37:56 David Jensen, a notary public contracted by Vail,
09:38:01 Christians and Associates, San Diego, California. This
09:38:02 video deposition is taking place at Cooley Godward,
09:38:07 Sacramento, and was noticed by Neil Kodsi of Womble,
09:38:12 Carlyle, et al.
09:38:13 Counsel, please voice-identify yourselves
09:38:14 and state whom you represent.
09:38:17 MR. KODSI: Neil Kodsi, representing the
09:38:20 RJ Reynolds Tobacco Company.
09:38:22 MS. MOORE: Alecia Moore, Paralegal with
Womble, Carlyle.
MR. CAFFERTY: Patrick Cafferty from Munger,
09:38:28 Tolles & Olson, representing Philip Morris.
09:38:32 MR. BRAUKMANN: Curtis Braukmann, with

09:38:33 Shook, Hardy & Bacon, representing Philip Morris.
09:38:36 MS. BRITTMAN: Shelley Brittman from
09:38:38 Sedgwick, Detert, Moran & Arnold, representing Brown &
09:38:39 Williamson.
09:38:40 MR. BROOKEY: Brian Brookey of Preston
09:38:42 Gates & Ellis, representing Plaintiff and People versus
09:38:45 Philip Morris and the American Environmental Safety
09:38:46 Institute.
09:38:48 THE VIDEOGRAPHER: Thank you.
09:38:48 The court reporter today is Shannon
09:38:50 Taylor-Scott of LegaLink. Would the reporter please
09:38:52 swear in the witness. Then we may begin.
09:39:02 (Whereupon, the witness was duly sworn.)

EXAMINATION BY MR. KODSI

09:39:02 MR. KODSI: Good morning, Dr. Pinkerton.
09:39:04 Let me introduce myself again on the record.
09:39:06 I'm Neil Kodsi, and I represent RJ Reynolds Tobacco
09:39:08 Company in the case caption that the videographer just

09:39:12 19 read. I won't repeat that lengthy caption, and we're
09:39:15 20 going to be asking you some questions today in your
09:39:17 21 deposition.
09:39:17 22 Q. Do you understand this is your deposition in
09:39:18 23 that case?
09:39:19 24 A. Yes.
09:39:20 25 Q. Okay. Have you been deposed before,
09:39:21 26 Dr. Pinkerton?
09:39:22 27 A. No.
09:39:22 28 Q. Okay. So this is your first time?

09:39:24 1 A. Yes.
09:39:25 2 Q. Let me just give you, what I generally do,
09:39:27 3 some instructions as to how the deposition is to
09:39:30 4 proceed and make sure that you're comfortable with
09:39:31 5 that. It being a deposition, although it's being
09:39:35 6 video'd, there is a court reporter taking it down, as
09:39:37 7 you see to your right, and she can't take down head
09:39:40 8 nods. So when I ask a question, if you could give a
09:39:43 9 verbal answer, that would be helpful. Although the
09:39:45 10 video can take down head nods, she won't be able to,
09:39:49 11 and I am going to be just asking you some questions
09:39:52 12 today.
09:39:52 13 If at any time I ask you a question that you
09:39:54 14 don't understand -- and I'm sure that will happen
09:39:56 15 throughout the day -- please feel free to tell me, and
09:39:58 16 we'll try to correct that understanding.
09:40:00 17 I'd ask that you don't speculate. If you
09:40:02 18 don't know the answer to a question, let me know before
09:40:05 19 you try to speculate as to the answer to a question.
09:40:07 20 If at any time today you feel like you need
09:40:09 21 a break, if you want -- if you need five minutes or ten
09:40:12 22 minutes, please let me know that, and we'll certainly
09:40:15 23 try to accommodate that as well.
09:40:16 24 Let me begin by asking if you brought any
09:40:20 25 documents with you today.
09:40:22 26 A. No.
09:40:25 27 Q. Okay. Have you been designated as an
09:40:27 28 "expert witness" in any other case?

09:40:29 1 A. No.
09:40:30 2 Q. How about as a "fact witness" in any case?
09:40:32 3 A. No.
09:40:34 4 Q. So this is actually the first case you've
09:40:36 5 ever -- or litigation you've ever been involved in?
09:40:39 6 A. Yes.
09:40:41 7 Q. Okay. Have you been asked to consult in any
09:40:44 8 other litigation?
09:40:45 9 A. No.
09:40:52 10 Q. Why don't we start by you telling me then
09:40:54 11 when you first got involved in this case that we're
09:40:56 12 here for today.
09:40:58 13 A. In December of 1998 or January of 1999.
09:41:05 14 Q. And I've said "this case" a few times. I
09:41:07 15 probably should have asked this up front.
09:41:08 16 What do you understand this case to be
09:41:09 17 about?
09:41:11 18 A. My understanding is that this is a trial to
09:41:16 19 work towards the banning of cigarette sales in the
09:41:20 20 State of California.
09:41:27 21 Q. Let me ask you to elaborate a little bit.

09:41:28 22 When you say, "work towards," what do you
09:41:30 23 mean by that?
09:41:31 24 A. That this will be a trial that will take
09:41:36 25 some time to process.
09:41:42 26 Q. And do you understand that the goal, the
09:41:43 27 Plaintiffs' goal in this case, is to ban the sale of
09:41:47 28 cigarettes in the State of California?

09:41:48 1 A. That is my understanding.
09:41:52 2 Q. Is that a goal that you agree with?
09:41:54 3 A. Yes.
09:42:06 4 Q. Do you think that cigarette sales should be
09:42:08 5 banned throughout the country?
09:42:10 6 A. Yes.
09:42:10 7 Q. And do you believe that cigarette sales
09:42:12 8 should be banned throughout the world?
09:42:15 9 A. Yes.
09:42:21 10 Q. And I'll assume this file -- I'll assume
09:42:23 11 then you believe that smoking should be banned
09:42:26 12 throughout the world as well.
09:42:27 13 A. Yes.
09:42:28 14 Q. Do you limit that to cigarettes, I mean
09:42:30 15 pipe, tobacco and everything else?
09:42:33 16 A. No, I don't limit it to just cigarettes.
09:42:38 17 Q. So you think all tobacco products that
09:42:41 18 emanate smoke should be banned throughout the world?
09:42:43 19 A. Yes.
09:42:46 20 Q. Okay. What do you know about the Plaintiffs
09:42:48 21 in this case?
09:42:57 22 A. Are you referring to Preston, Gates & Ellis?
09:43:00 23 Q. Well, that's the Plaintiffs' law firm.
09:43:01 24 Do you know who the actual Plaintiffs are
09:43:03 25 who have brought this lawsuit?
09:43:05 26 A. No.
09:43:06 27 Q. Are you familiar with the -- and correct me
09:43:09 28 if I say this wrong -- the American Environmental

09:43:12 1 Safety Institute?
09:43:13 2 A. No.
09:43:14 3 Q. Are you familiar with them?
09:43:15 4 A. (Shakes head.)
09:43:16 5 Q. Okay. You haven't met with anyone from that
09:43:17 6 organization then?
09:43:18 7 A. No.
09:43:19 8 Q. Okay. How have you obtained your
09:43:20 9 information about this case?
09:43:22 10 A. Through meeting with Mr. Carrick.
09:43:34 11 Q. And Mr. Carrick is an attorney with Preston,
09:43:34 12 Gates & Ellis?
09:43:34 13 A. Yes.
09:43:34 14 Q. Who else have you met with regarding this
09:43:34 15 case?
09:43:35 16 A. One of his associates, actually, Carolyn
09:43:41 17 Sieve.
09:43:42 18 Q. Okay. And have you also met with
09:43:43 19 Mr. Brookey?
09:43:45 20 A. Yes.
09:43:45 21 Q. Is there anyone else you've met with
09:43:47 22 regarding this case?
09:43:48 23 A. Yes. Mr. McGuire and Mr. Hulburt.
09:43:59 24 Q. McGuire. Would that be Mickey McGuire?

09:44:02 25 A. Yes.
09:44:02 26 Q. It would be the Thorsnes law firm?
09:44:05 27 A. Yes.
09:44:05 28 Q. And who was the other name?

09:44:07 1 A. Chris Hulburt.
09:44:08 2 Q. Is Chris Hulburt also with Mr. McGuire's
09:44:11 3 firm?
09:44:12 4 A. Yes.
09:44:13 5 Q. Do you know if he is an attorney?
09:44:17 6 A. I am not sure.
09:44:18 7 Q. Actually, you said, "Chris." I assume --
09:44:20 8 Chris is a male?
09:44:21 9 A. Yes.
09:44:21 10 Q. Okay.
09:44:22 11 A. Uh-huh. Uh-huh.
09:44:24 12 Q. These days, you never know.
09:44:27 13 Why don't you describe for me -- we talked
09:44:32 14 about you first being involved in this case either in
09:44:34 15 December of '98 or January of '99, how you were first
09:44:38 16 approached or how you first got involved in the case.
09:44:41 17 A. My recollection is that I received a phone
09:44:45 18 call from Mr. Carrick. He was interested in the
09:44:51 19 research that we had done, and he explained to me that
09:44:59 20 there was a potential trial that was going to go
09:45:04 21 forward having to do with Proposition 65 for the State
09:45:10 22 of California and that he was inquiring as to whether I
09:45:15 23 would be interested or willing to serve as an expert
09:45:19 24 witness regarding the health effects of environmental
09:45:24 25 tobacco smoke in humans as we study it using animal
09:45:31 26 models.
09:45:37 27 Q. And what was your response to him at that
09:45:39 28 time if you remember?

09:45:41 1 A. At first, I was a bit reluctant because I
09:45:44 2 didn't understand what that might entail. So he gave
09:45:50 3 me some further explanations, and I eventually agreed
09:45:57 4 to meet with him and to discuss the sorts of things
09:46:02 5 that they needed to know of me, whether I would be
09:46:09 6 involved in such a case or not.
09:46:12 7 Q. What sorts of things did you think were
09:46:14 8 important that they needed to know about you?
09:46:16 9 (Mr. Lendrum joins the proceedings.)
09:46:19 10 THE WITNESS: Well, basically the types of
09:46:22 11 studies that we did, whether they would be relevant to
09:46:27 12 the kind of information that would be needed to
09:46:34 13 establish whether there's a health effect associated
09:46:39 14 with exposure to environmental tobacco smoke and the
09:46:42 15 relevance of animal studies to be able to determine
09:46:48 16 what health effects might be associated with exposures
09:46:52 17 and what kind of conditions might occur in terms of
09:46:58 18 changes in the -- especially in the respiratory system.
09:47:04 19 MR. KODSI: Q. And how did you describe to
09:47:05 20 them what you thought the relevance of the animal
09:47:07 21 studies would be to that conclusion?
09:47:12 22 A. That animal studies, I felt, are -- were
09:47:17 23 very relevant to what has been seen epidemiologically
09:47:23 24 in human studies. Many of the findings that have --
09:47:28 25 have been deduced by epidemiological studies have been
09:47:33 26 confirmed by animal studies.
09:47:46 27 Q. Okay. At that point, let's still -- and

09:47:47 28 we'll talk about that a lot in a lot greater detail.

09:47:50 1 I want to go back to talking about your
09:47:52 2 discussions with Mr. Carrick. At that point in time,
09:47:56 3 did you understand that there was a role for you in
09:47:57 4 this case or what your role was to be in this case?
09:48:05 5 A. Yes, that I would be someone involved in
09:48:11 6 basic science research, having to do with actual
09:48:17 7 exposures to environmental tobacco smoke under
09:48:22 8 experimental conditions and that I would report on our
09:48:27 9 findings and the interpretation of our findings for
09:48:31 10 those studies.
09:48:34 11 Q. Do you understand your role in this case
09:48:35 12 then to talk about the animal experiments that you've
09:48:42 13 conducted?
09:48:42 14 A. I think that it covers more than that.
09:48:44 15 Q. Okay. Could you expand? Maybe I didn't
09:48:46 16 understand. What beyond that?
09:48:48 17 A. Uh-huh. I think the interpretation of
09:48:53 18 changes during lung development that would be
09:48:56 19 associated with exposure to cigarette smoke.
09:49:04 20 The -- from an anatomical perspective, from
09:49:08 21 a structural perspective, from functional changes that
09:49:12 22 might be occurring in the respiratory system as well as
09:49:20 23 a characterization of the type of exposure conditions
09:49:25 24 that lead to such changes.
09:49:29 25 Also the importance of timing of exposure
09:49:37 26 that would include critical windows of exposure during
09:49:42 27 perinatal development and to interpret these findings
09:49:50 28 and extrapolate those to human conditions, whether

09:49:57 1 there were similarities or dissimilarities in the types
09:50:00 2 of findings that we found in our animal studies
09:50:06 3 compared to studies that have been reported in the peer
09:50:08 4 reviewed literature.
09:50:12 5 Q. When you say characteristics of exposure
09:50:15 6 conditions, what do you mean by characteristics of
09:50:17 7 exposure conditions?
09:50:18 8 A. For our animal studies, it was very
09:50:21 9 important to us to be sure that we knew exactly the
09:50:24 10 conditions we were exposing animals to so that they
09:50:28 11 would be reproducible and so that they could be
09:50:33 12 reconfirmed either by repetition -- repeated studies in
09:50:39 13 our laboratory or in someone else's laboratory, as long
09:50:43 14 as they knew exactly how we did those exposures so that
09:50:51 15 they would be valid, validated in that manner.
09:50:56 16 Q. When you talk about characteristics of
09:50:58 17 exposure conditions, you're talking about the animal
09:51:00 18 exposures, not human exposures in the real world?
09:51:03 19 A. For my own studies, yes.
09:51:06 20 Q. Do you intend to testify in this case as an
09:51:09 21 expert about human exposures in the real world?
09:51:17 22 A. From the perspective that the epidemiology
09:51:17 23 and the studies that are out in the literature are
09:51:20 24 important to better understanding our animal inhalation
09:51:24 25 studies.
09:51:26 26 Q. And we've talked a little bit about
09:51:28 27 epidemiology, and we'll go into that in greater detail
09:51:32 28 later, but let me go ahead and understand now:

09:51:34 1 Do you intend to testify in this case about
09:51:40 2 the epidemiology of ETS, the epidemiology studies on
09:51:40 3 ETS?
09:51:41 4 A. From the perspective that I understand the
09:51:44 5 reading of them.
09:51:45 6 Q. Do you view yourself as an expert in
09:51:47 7 epidemiology?
09:51:48 8 MR. BROOKEY: I object to the extent it
09:51:49 9 calls for a legal conclusion, but he can answer.
09:51:51 10 THE WITNESS: From the perspective that it
09:51:53 11 is very important for me to understand epidemiology in
09:51:56 12 order to design experiments for animal studies.
09:52:13 13 MR. KODSI: Q. Dr. Pinkerton, how many
09:52:14 14 hours have you spent working on this case?
09:52:18 15 A. Approximately 35 hours.
09:52:25 16 Q. Do you have a written breakdown of what that
09:52:28 17 35 hours has entailed?
09:52:32 18 A. From the perspective of billing?
09:52:35 19 Q. Yes.
09:52:36 20 A. No.
09:52:37 21 Q. All right. Could you describe generally
09:52:39 22 what you've done over the course of that 35 hours?
09:52:42 23 A. Yes. Approximately five to six hours were
09:52:48 24 in meeting with Doctor -- with Mr. Carrick, and 30
09:52:58 25 hours was in the preparation of the declaration which I
09:53:03 26 submitted to Preston, Gates & Ellis. This included
09:53:09 27 literature review, interpretation of a number of
09:53:16 28 studies, a summarizing of our own research and writing

09:53:23 1 up drafts of the declaration.
09:53:28 2 Q. Now, did the 30 hours of preparing the
09:53:31 3 declaration all occur after your five to six hours of
09:53:34 4 meeting with Mr. Carrick?
09:53:36 5 A. Yes.
09:53:37 6 Q. So you filed your declaration in this case
09:53:41 7 October of '99 -- is that correct? -- give or take a
09:53:46 8 month?
09:53:47 9 A. Yes.
09:53:48 10 Q. So have you not -- have you spent any time
09:53:50 11 working on this case since October of '99?
09:53:53 12 A. No.
09:53:55 13 Q. Have you had any meetings with any of the
09:53:56 14 attorneys we talked about earlier since October of '99?
09:54:03 15 A. Only with Mr. McGuire, Hulburt and
09:54:08 16 Mr. Brookey.
09:54:09 17 Q. And how long were those meetings?
09:54:11 18 A. Approximately four hours.
09:54:18 19 Q. And those aren't included in the 35 that we
09:54:21 20 just talked about?
09:54:22 21 A. No.
09:54:23 22 Q. Do you intend on billing them for that time?
09:54:26 23 A. Yes.
09:54:32 24 Q. And what is your compensation rate in this
09:54:34 25 case?
09:54:35 26 A. $200 per hour.
09:54:45 27 Q. Now, we talked about other litigation you
09:54:49 28 may have been involved in.

09:54:50 1 Have you ever served as a consultant before
09:54:52 2 or is this the only case you're doing that in?
09:54:55 3 MR. BROOKEY: I'll object. That's been

09:54:56 4 asked and answered. He can answer it again.
09:54:58 5 MR. KODSI: Let me rephrase that because
09:55:00 6 Brian's right because I don't want it to be the same
09:55:03 7 question asked before.
09:55:04 8 Q. What I guess I'm asking is, unrelated to
09:55:06 9 litigation, have you ever served as a consultant?
09:55:08 10 A. Yes.
09:55:09 11 Q. Is $200 an hour the rate you charge for
09:55:13 12 those consulting services as well?
09:55:19 13 A. I don't recall. I don't think I charged on
09:55:23 14 an hourly basis.
09:55:26 15 Q. Are you at liberty to describe the
09:55:28 16 consulting services you've done? I don't want to get
09:55:30 17 into anything that may be privileged.
09:55:36 18 A. Yes. I consulted with Schering Plough on
09:55:40 19 aspects of lung growth and development.
09:55:45 20 Q. And who was that?
09:55:47 21 A. Schering Plough.
09:55:48 22 Q. Is that a person or --
09:55:49 23 A. It's a pharmaceutical company.
09:55:52 24 Q. Okay. It's not one I'm familiar with.
09:55:54 25 A. Uh-huh.
09:55:54 26 Q. And who else?
09:55:56 27 A. That's all.
09:56:06 28 Q. Okay. Why don't we talk about, briefly, the

09:56:08 1 five to six hours where you met with Mr. Carrick before
09:56:12 2 your declaration in this case. If you could just
09:56:14 3 generally describe for me what was discussed during
09:56:16 4 that five to six hours.
09:56:19 5 A. A review of the research that I had been
09:56:24 6 instrumental in doing, so I provided to them copies of
09:56:32 7 each of our peer reviewed publications, and these were
09:56:37 8 discussed in a general manner for them to better
09:56:41 9 understand the results and the findings from those
09:56:48 10 studies.
09:56:48 11 There was also some discussion about
09:56:52 12 exposure assessment and questions about information
09:57:01 13 that had actually been prepared previously by another
09:57:07 14 consultant of theirs.
09:57:10 15 Q. Which consultant would that be?
09:57:12 16 A. Dr. Nazaroff.
09:57:17 17 Q. Do you remember what you discussed about
09:57:18 18 Dr. Nazaroff?
09:57:21 19 A. Actually, that -- it was just simply
09:57:25 20 reviewing some of the studies that he had done on
09:57:28 21 exposure assessment, and...
09:57:34 22 Q. Were you familiar with Dr. Nazaroff prior to
09:57:36 23 your meeting with Mr. Carrick?
09:57:39 24 A. Only in name only.
09:57:43 25 Q. Now, you said you spent approximately 30
09:57:45 26 hours preparing your declaration in this case.
09:57:49 27 Could you describe generally what you did
09:57:50 28 during that 30 hours to prepare the declaration?

09:57:56 1 A. It was primarily a review of the literature
09:57:59 2 having to do with environmental tobacco smoke and
09:58:06 3 exposures during perinatal development. So this
09:58:11 4 literature review primarily focused on children and
09:58:15 5 health effects associated with exposure of children to
09:58:19 6 environmental tobacco smoke, also a general review of

09:58:26 7 our own research studies that we had conducted over
09:58:30 8 approximately the previous eight to nine years, and a
09:58:38 9 preparation of -- and summary of the -- those research
09:58:45 10 studies into a written document.
09:58:50 11 Q. And you mentioned four hours that you spent
09:58:52 12 meeting with Mr. McGuire. When did -- is that one
09:58:55 13 four-hour meeting or a series of meetings encompassing
09:58:59 14 four hours?
09:59:00 15 A. No. Actually, three hours with Mr. McGuire
09:59:04 16 and one hour with Mr. Brookey.
09:59:12 17 Q. Okay. When was the three-hour meeting with
09:59:13 18 Mr. McGuire?
09:59:14 19 A. Last week.
09:59:21 20 Q. What did you all discuss there?
09:59:21 21 A. What entails a deposition, just general
09:59:24 22 information about the sorts of things that I could
09:59:29 23 possibly expect in terms of what happens in a
09:59:34 24 deposition, the fact that there is a videotaping of the
09:59:40 25 deposition and a transcript of all the discussions at a
09:59:46 26 deposition.
09:59:48 27 Q. Anything else?
09:59:52 28 A. A clarification of how this -- the -- of how

10:00:00 1 things have changed, that this no longer is a Prop 65
10:00:04 2 issue.
10:00:06 3 Q. Did you go over any documents during that
10:00:07 4 three-hour meeting?
10:00:11 5 A. Yes. My declaration. We talked a bit about
10:00:19 6 some of the terminology, the verbiage that I had used
10:00:23 7 in the declaration, and they asked for clarification.
10:00:30 8 Q. Did you take any notes on your declaration
10:00:33 9 or otherwise during that meeting?
10:00:36 10 A. No.
10:00:37 11 Q. All right. Do you remember specifically --
10:00:39 12 and we'll be going through your declaration a little
10:00:41 13 bit later. Do you remember specifically anything you
10:00:43 14 discussed about your declaration during that meeting?
10:00:47 15 A. Primarily the fact that there were certain
10:00:50 16 terms that I used that were -- that were questioned,
10:00:58 17 the use of "suggests," "probability," "maybe," certain
10:01:05 18 terms like that. The discussion was whether those were
10:01:13 19 scientifically plausible or was it actually a fact that
10:01:20 20 that was what could happen.
10:01:22 21 Q. So they asked you to interpret terms like
10:01:25 22 "suggests," "probably" and "maybe"?
10:01:27 23 A. Right.
10:01:29 24 Q. Okay. Well, maybe when we go through the
10:01:30 25 declaration, we might try to point some of those out
10:01:32 26 and talk through them.
10:01:35 27 Is there anyone else that's associated with
10:01:40 28 you, maybe in your office or at the University, that's

10:01:43 1 working with you on this case?
10:01:45 2 A. No.
10:01:49 3 Q. Do you feel that you have any additional
10:01:50 4 work that you need to do before you'll be ready to
10:01:52 5 testify in this case?
10:01:53 6 A. No.
10:01:56 7 Q. We've talked about the attorneys you've
10:01:59 8 spoken with related to this case.
10:02:01 9 Have you spoken with any of the experts in

this case? I know you work with a few of them.
A. Yes.
Q. So why don't we just ask that question generally; then maybe I'll ask specifically, but is there generally anyone you've spoken with about this case other than the attorneys?
A. Yes. Dr. Witschi --
Q. Right.
A. -- and Dr. Joad.
Q. Have you spoken with Dr. Slotkin?
A. Not about this case.
Q. Did you know Dr. Slotkin prior to your involvement in this case?
A. Yes.
Q. All right. And how did you know Dr. Slotkin?
A. He taught me in a course while I was a student at Duke University.
Q. Which course was that?

A. A course in pharmacology.
Q. Have you all corresponded since that course?
A. Yes.
Q. About any tobacco-related issues?
A. Yes.
Q. Could you generally describe what you've talked about as far as it relates to tobacco issues?
A. We have established a collaborative effort in research to examine the effects of tobacco smoke on nonhuman primates as well as on rodents, and we are in the process of trying to finalize our first experiment.
Q. When did that relationship begin as far as a collaborative effort to research tobacco?
A. In, probably, June of 1999.
Q. Now, is that in any way related to this case?
A. No.
Q. Did you correspond with Dr. Slotkin at all prior to December of '98 other than having him teach you in a course at Duke?
A. No.
Q. Is the fact that you and he decided to work together in any way related to the fact that you're both involved with this case?
A. No.
Q. Okay. We've talked about discussions you've had with Drs. Witschi and Joad, and I mentioned Slotkin. What about Dr. Nazaroff?

A. Yes.
Q. Is there anybody else that you've talked about this case with?
A. No.
Q. Why don't we start each one of those one at a time. Why don't we start with Dr. Witschi.
A. Our discussions have simply been that we're both -- we're going to go -- undergo depositions.
Q. Have you talked to Dr. Witschi about his deposition?
A. No.
Q. Is there any work that you and Dr. Witschi

10:05:04 13 are doing together to prepare for this case?
10:05:06 14 A. No.
10:05:08 15 Q. What about Dr. Joad?
10:05:13 16 A. Same arrangement. We've simply talked about
10:05:16 17 the fact that we both have depositions.
10:05:20 18 Q. And are you and Dr. Joad in any way working
10:05:22 19 together to help prepare you to testify in this case?
10:05:28 20 A. No, but I know that her declaration covers
10:05:34 21 the clinical relevance of exposure to environmental
10:05:38 22 tobacco smoke while mine covers the relevance of
10:05:43 23 environmental tobacco smoke and exposure to animal
10:05:46 24 models and how we interpret that and extrapolate that
10:05:52 25 to human conditions.
10:05:55 26 Q. Yes. I was going to ask you that.
10:05:56 27 Did you and Dr. Joad at all work together in
10:05:59 28 preparing each of your separate declarations?

10:06:01 1 A. No.
10:06:03 2 Q. Did you share any drafts with Drs. Joad or
10:06:08 3 Witschi of your declaration?
10:06:10 4 A. No.
10:06:10 5 Q. And did they share any of their drafts with
10:06:13 6 you?
10:06:13 7 A. Yes.
10:06:14 8 Q. Both of them or --
10:06:16 9 A. No. Dr. Joad.
10:06:17 10 Q. Okay. Did you give her comments back on her
10:06:20 11 draft?
10:06:21 12 A. No.
10:06:22 13 Q. Neither orally nor verbally -- I mean
10:06:25 14 neither verbally or in writing?
10:06:27 15 A. That's correct.
10:06:32 16 Q. Okay. And that leaves us with Doctor -- and
10:06:34 17 I believe it's pronounced Naz-aur-off, not Naza-roff,
10:06:38 18 so I may have been mispronouncing it earlier.
10:06:41 19 Dr. Nazaroff, have you spoken with him about
10:06:43 20 this case?
10:06:46 21 A. Only from the exposure assessment.
10:06:51 22 So it's -- no, not in directly discussing
10:06:54 23 the case with him, but it was in connection with the
10:07:01 24 case that I met with him with Mr. Carrick.
10:07:09 25 Q. You had a meeting with Dr. Nazaroff and
10:07:09 26 Mr. Carrick?
10:07:10 27 A. Yes.
10:07:10 28 Q. When did that take place?

10:07:12 1 A. April 19th of 1999.
10:07:20 2 Q. And you may have already answered this, but
10:07:23 3 just to make sure: Could you describe generally what
10:07:26 4 you discussed at that meeting unless it's something
10:07:28 5 you've already explained to me?
10:07:31 6 A. Basically, exposure assessments in -- in
10:07:40 7 field studies, what constitutes an exposure. There
10:07:46 8 were some discussions about if animal studies help to
10:07:52 9 clarify a dose response relationship in exposure
10:07:58 10 assessments, the validity of looking at carcinogenic
10:08:06 11 effects or looking at non carcinogenic effects.
10:08:14 12 Q. The validity of looking -- when you say,
10:08:15 13 "the validity of looking at carcinogenic effects," what
10:08:18 14 do you mean by that?
10:08:20 15 A. That whether animal models would be

10:08:24 16 appropriate for studying the carcinogenic effects of
10:08:30 17 environmental tobacco smoke and whether, in a similar
10:08:36 18 manner, the use of animal models would be appropriate
10:08:39 19 for understanding health effects of environmental
10:08:42 20 tobacco smoke as seen in humans.
10:08:46 21 Q. Now, do you view Dr. Nazaroff as an expert
10:08:48 22 in animal studies?
10:08:55 23 A. I don't know the answer to that.
10:08:58 24 Q. Were you the one mainly talking about animal
10:09:00 25 studies in that conversation?
10:09:02 26 A. Yes.
10:09:03 27 Q. What area of expertise do you view
10:09:04 28 Dr. Nazaroff to have? What do you think his area of

10:09:08 1 expertise is?
10:09:09 2 MR. BROOKEY: Again, I'll object to the
10:09:10 3 extent it calls for a legal conclusion and for
10:09:13 4 speculation, but he may answer.
10:09:14 5 THE WITNESS: My understanding is that he
10:09:16 6 has expertise in air quality monitoring and exposure
10:09:22 7 assessments.
10:09:23 8 MR. KODSI: Q. Do you view Dr. Nazaroff as
10:09:25 9 having expertise in ETS exposures?
10:09:28 10 MR. BROOKEY: Same objections.
10:09:30 11 THE WITNESS: My opinion would be yes.
10:09:33 12 MR. KODSI: Q. What about ETS chemistry?
10:09:35 13 MR. BROOKEY: Same objections.
10:09:40 14 THE WITNESS: I don't know, but I would
10:09:41 15 assume so based on his studies.
10:09:43 16 MR. KODSI: Q. But you would be speculating
10:09:45 17 there, you would agree? When you say, "I assume so," I
10:09:47 18 assume that means that you're really not sure.
10:09:50 19 A. That's correct.
10:10:02 20 Q. Okay. Have you done anything specifically
10:10:04 21 to prepare for this deposition, Dr. Pinkerton?
10:10:08 22 A. Yes.
10:10:09 23 Q. Could you describe that for me?
10:10:12 24 A. I've read my declaration again. I've
10:10:15 25 reviewed a number of the references that are
10:10:21 26 pertinent -- that I'm a coauthor and author on. I've
10:10:29 27 read some of the chapters in the California
10:10:35 28 Environmental Protection Agency book on environmental

10:10:39 1 tobacco smoke, and I have also briefly reviewed the
10:10:51 2 deposition transcript of Dr. Slotkin and briefly the
10:10:58 3 deposition transcript of Dr. Witschi.
10:11:06 4 Q. Why did you briefly review the deposition
10:11:09 5 transcript of Dr. Slotkin?
10:11:13 6 A. It was given to me by Mr. McGuire to show me
10:11:19 7 what happens in a deposition, the type of questions
10:11:25 8 that may be asked, the types of responses that may be
10:11:28 9 given.
10:11:29 10 Q. Was there anything in there he asked you to
10:11:31 11 pay particular attention to?
10:11:32 12 A. No.
10:11:33 13 Q. Was the copy that he gave you highlighted or
10:11:35 14 marked up in any way?
10:11:36 15 A. No.
10:11:38 16 Q. Okay. Did you highlight or mark up the copy
10:11:39 17 that you were given?
10:11:40 18 A. No.

10:11:44 19 Q. Now, you also said you reviewed the
10:11:46 20 deposition of Dr. Witschi. Why did you feel it was
10:11:48 21 important to review that deposition transcript?
10:11:51 22 A. It was given to me, and although I didn't
10:11:54 23 have much time to look at it, I did briefly review a
10:12:02 24 few pages of the deposition.
10:12:04 25 Q. Now, his deposition was taken, I think, on
10:12:07 26 April 10th and then at some point within the last few
10:12:10 27 weeks. Did you review the April 10th transcript, the
10:12:14 28 more recent transcript or both?

10:12:16 1 A. My understanding is that it's the April 10th
10:12:20 2 transcript.
10:12:21 3 Q. Okay. And when you say it was given to you,
10:12:22 4 I assume that it was also given to you by Mr. McGuire?
10:12:25 5 A. That's correct.
10:12:25 6 Q. Okay. And did he mark up or ask you to pay
10:12:28 7 any particular attention to anything in that
10:12:30 8 transcript?
10:12:31 9 A. No.
10:12:31 10 Q. Okay. And, in your copy of the transcript,
10:12:34 11 are there any highlights or notes?
10:12:36 12 A. No.
10:12:38 13 Q. And I guess I should ask for both Slotkin
10:12:40 14 and Witschi. Did you take any notes on separate sheets
10:12:43 15 of paper regarding those depositions?
10:12:45 16 A. No.
10:12:48 17 MR. KODSI: Let's go ahead and mark that. I
10:12:55 18 only brought two copies of each. This is just the
10:12:57 19 expert declaration, Brian, if we could.
20 (Whereupon, Defendants' Exhibit 527 was
21 marked for identification.)
10:13:50 22 MR. KODSI: Q. Okay. Dr. Pinkerton, you've
10:13:51 23 been handed what has been marked as Exhibit 527. Just
10:13:57 24 looking at the -- if you need time to put your glasses
10:14:00 25 on, you sure can. Just looking at the cover page, is
10:14:02 26 that a document that you recognize?
10:14:06 27 A. Yes.
10:14:08 28 Q. Okay. And I have just copied page -- the

10:14:13 1 cover and then Page 7, if you'd flip to what's marked
10:14:17 2 as Page 7, it's really Page 3 of the exhibit.
10:14:21 3 Do you recognize that description of your
10:14:23 4 testimony there?
10:14:25 5 A. Yes.
10:14:26 6 Q. Okay. This is something you've seen before?
10:14:28 7 A. Yes.
10:14:29 8 Q. When did you first see this document, if you
10:14:31 9 remember?
10:14:38 10 A. I know I saw it last week.
10:14:44 11 Q. Now what do you understand that description
10:14:46 12 to be?
10:14:51 13 A. Of the areas and topics that I would testify
10:14:54 14 as, as an expert witness, if this should go to trial.
10:14:59 15 Q. Did you assist in the preparation of that
10:15:01 16 document?
10:15:02 17 A. No.
10:15:03 18 Q. Do you know how it was prepared?
10:15:07 19 A. No.
10:15:08 20 Q. It was filed in -- you know, I thought -- I
10:15:14 21 don't have the date. It was filed at some point last

10:15:16 22 year in 1999. Is that -- did you see it before it was
10:15:19 23 filed?
10:15:20 24 A. No.
10:15:22 25 Q. Okay. If you'll turn to where it has your
10:15:25 26 name on Page 7, it indicates that you're going to
10:15:30 27 testify regarding the effects of prenatal and postnatal
10:15:34 28 exposure to ETS on lung growth and development as

10:15:44 1 evidenced by animal studies.
10:15:44 2 Is that an accurate representation of what
10:15:44 3 you intend to testify about at this trial?
10:15:46 4 A. Yes.
10:15:49 5 Q. Is your testimony limited to the effects
10:15:52 6 evidenced by animal studies?
10:15:54 7 A. No.
10:15:55 8 Q. How would you expand it beyond what it says
10:15:57 9 there?
10:16:00 10 A. I would -- I would state that we can use
10:16:04 11 animal studies to confirm health effects also seen in
10:16:11 12 humans and potential mechanisms by which those health
10:16:17 13 effects are manifested in humans.
10:16:25 14 Q. "We can use animal studies."
10:16:27 15 When you say, "We can use animal studies to
10:16:29 16 confirm health effects... seen in humans," is it your
10:16:32 17 opinion that animal studies alone cannot demonstrate
10:16:37 18 causation in humans?
10:16:39 19 A. I --
10:16:40 20 MR. BROOKEY: I'm sorry. I'll object to the
10:16:41 21 extent it calls for a legal conclusion and it's vague
10:16:44 22 and ambiguous, but he can answer.
10:16:46 23 THE WITNESS: I would think that animal
10:16:47 24 studies simply confirm what is already known in human
10:16:52 25 studies.
10:16:53 26 MR. KODSI: Q. I guess what I'm trying to
10:16:54 27 understand is, if we assume for a given disease end
10:16:58 28 point that we don't have human studies, can animal

10:17:02 1 studies by themselves demonstrate causation in humans?
10:17:07 2 MR. BROOKEY: Objection. It still calls for
10:17:09 3 a legal conclusion; it's an incomplete hypothetical,
10:17:13 4 lacks foundation, but he can answer.
10:17:16 5 THE WITNESS: I think the value of animal
10:17:17 6 studies is the fact that we can control precisely
10:17:21 7 exposure conditions which we can't with human
10:17:26 8 conditions and that we can also look more precisely in
10:17:32 9 potential relationships of alterations in the structure
10:17:39 10 or function of the respiratory system based on our
10:17:43 11 exposure conditions that are not possible in human
10:17:48 12 studies.
10:17:49 13 MR. KODSI: Q. Do you intend to testify
10:17:50 14 about the human study, any human studies to this trial?
10:17:55 15 A. Only as it relates to our animal studies.
10:18:00 16 Q. I guess I'm trying to understand what you
10:18:01 17 mean by that. Are you going to interpret any
10:18:08 18 epidemiology studies at the trial?
10:18:13 19 A. Only from the perspective that
10:18:17 20 epidemiological studies give us the direction that we
10:18:21 21 need to have in order to perform animal studies to have
10:18:25 22 them be valid in terms of a scientific reason for
10:18:35 23 saying that environmental tobacco smoke may cause
10:18:38 24 "this" type of health effect or "that" type of

10:18:41 25 alteration and lung function.
10:18:45 26 Q. In other words, you view the epidemiology
10:18:47 27 studies as helping to provide you with the basis for
10:18:55 28 your animal studies?

10:18:58 1 A. That is correct.
10:18:59 2 Q. Do you view yourself as having expertise in
10:19:02 3 interpreting the validity of epidemiology studies?
10:19:06 4 MR. BROOKEY: Objection to the extent it
10:19:07 5 calls for a legal conclusion, but he can answer.
10:19:10 6 THE WITNESS: From the perspective of my
10:19:13 7 training as a scientist and the need to have a good
10:19:19 8 grasp of epidemiology in order to formulate a research
10:19:25 9 plan for doing studies to confirm if we see similar
10:19:31 10 effects in animals, yes.
10:19:44 11 MR. KODSI: Q. Okay. Let's go back to this
10:19:45 12 exhibit, Number 527, and I want to ask you if you could
10:19:50 13 define -- you talk about the effects of prenatal and
10:19:54 14 postnatal exposure to ETS. Now, these are going to
10:19:58 15 seem very simplistic, but I just want to get the
10:20:00 16 definitions on the record, if you could define for me
10:20:03 17 what you mean by "prenatal."
10:20:06 18 A. These would be exposures that would occur
10:20:09 19 during the gestational period. So they would actually
10:20:12 20 be maternal exposure to environmental tobacco smoke
10:20:17 21 during pregnancy. That would be the definition for
10:20:23 22 "prenatal exposure."
10:20:25 23 Q. Now, when we talk about maternal exposure to
10:20:28 24 ETS during pregnancy, do you intend to testify about
10:20:33 25 effects on the mother, the infant still inside the
10:20:40 26 mother or both?
10:20:41 27 A. On the infant.
10:20:47 28 Q. Okay.

10:20:47 1 A. It would be best to say that that would be
10:20:49 2 the effects on the fetus.
10:20:52 3 Q. And do you intend to testify about maternal
10:20:56 4 exposure to ETS, the effects of maternal exposure to
10:21:00 5 ETS, maternal smoking or both?
10:21:04 6 A. Maternal exposure to ETS.
10:21:06 7 Q. So you do not intend to testify about any
10:21:09 8 effects of actual maternal smoking on the unborn child?
10:21:13 9 A. Not from the animal model studies that we're
10:21:18 10 doing.
10:21:19 11 Q. Now, if you could now define the "postnatal
10:21:23 12 period" for me.
10:21:26 13 A. "Postnatal period" refers to any exposures
10:21:29 14 that occur from birth through adulthood.
10:21:39 15 Q. I guess when you say, "through adulthood,"
10:21:41 16 how do you define "adulthood"?
10:21:44 17 A. In an animal study, we typically consider a
10:21:50 18 number of factors. One would be reaching sexual
10:21:53 19 maturity. Another would also be reaching a plateau in
10:21:59 20 terms of lung growth, that there is no further
10:22:06 21 formation of alveoli within the gas exchange regions of
10:22:11 22 the lung and that the expansion of the lungs is slowing
10:22:18 23 down.
10:22:21 24 In rodents, they don't ever totally stop
10:22:25 25 developing in terms of the size of the lung, but
10:22:30 26 typically, we would consider an adult -- adulthood in
10:22:35 27 an animal to be one where they've reached sexual

maturity and, two, that they have come to a plateau in

terms of having further growth.

Q. Do you differentiate between the point in time where an animal reaches adulthood and a point in time in which a human reaches adulthood?

A. We think that there are correlations that we can follow. Usually, an animal will stop the process of alveolarization around 3 to 4 weeks of age. In a human, alveolarization is thought to end between 2 years and 6 years of age.

Q. Now, do you intend to testify about the effects of ETS on adults?

A. Only from the perspective that -- if our studies of exposure to ETS continued through the adulthood of the animal.

Q. Do you have studies on exposure of ETS that continue through the adulthood of the animal?

A. Yes.

Q. And what end points would those study?

A. Lung function, the lung structure and lung biochemistry.

Q. Is there anything else you intend to testify about that we haven't just discussed?

A. Not that I can think of at the moment.

Q. Doctor, let's start broad and we'll work our way narrow. It says that you're going to testify regarding the effects of prenatal and postnatal exposure to ETS on lung growth and development.

Why don't you begin by just explaining to me

what are the effects of ETS exposure -- or prenatal and postnatal ETS exposure on lung growth and development.

A. Uh-huh. Well, I could begin by just talking about the prenatal effects.

Q. Why don't we separate that out. That's a good suggestion. Why don't we start out with what the prenatal effects are.

A. From our studies, we have found that prenatal exposure to environmental tobacco smoke leads to a small but significant reduction in fetal body weight that is measured near the completion of gestation or at term. There is also studies that we've completed to show that there is a change in the maturation of fetal epithelial cells lining the airways with maternal exposure to environmental tobacco smoke.

From a postnatal perspective, we have found and I would testify that there are developmental alterations with exposure to environmental tobacco smoke that include changes in the maturation of the lung postnatally so that the gas exchange portions develop in a different pattern during this postnatal period of development, including changes in tissue compartment volumes as well as alveolar surface area for gas exchange, that there are also alterations in the metabolic profile that develops postnatally in all the mammalian respiratory systems, that we see that environment -- exposure during the postnatal period to environmental tobacco smoke leads to a change in the

10:27:10 1 way cells express metabolic enzymes that are important
10:27:15 2 in processes of metabolizing foreign materials that
10:27:22 3 enter into the respiratory system, that these change
10:27:25 4 very early during postnatal development and that they
10:27:29 5 are sustained with continued exposure to environmental
10:27:34 6 tobacco smoke, that there is no adaptive response that
10:27:41 7 brings back these values -- or these metabolic
10:27:45 8 functions to control values during periods of exposure
10:27:50 9 to environmental tobacco smoke.
10:28:00 10 I will also testify about the changes that
10:28:02 11 are seen with exposure during very critical windows of
10:28:09 12 lung development that lead to significant changes in
10:28:13 13 lung function and airway reactivity based on studies to
10:28:21 14 look at airway function and challenge with
10:28:26 15 pharmacological agents that are used to look at lung
10:28:31 16 function in an airway reactivity in the respiratory
10:28:38 17 system over a wide range of mammalian species, but of
10:28:44 18 course, I'll just be talking about it in rats.
10:28:49 19 Q. Now, why would you just be talking about it
10:28:51 20 in rats?
10:28:52 21 A. Because that's the animal model that we've
10:28:55 22 used for --
10:28:56 23 Q. Okay.
10:28:56 24 A. -- a number of these studies.
10:28:58 25 Q. Now, for all of the things that we've just
10:29:01 26 talked about, that we've just discussed, are you going
10:29:03 27 to be limiting your testimony to the studies that
10:29:05 28 you've conducted?

10:29:06 1 A. No, not necessarily, but at this point, I'm
10:29:10 2 just telling you what findings that we found --
10:29:14 3 Q. Okay.
10:29:15 4 A. -- in our own studies, and also the fact
10:29:20 5 that there are changes that occur within rare
10:29:27 6 epithelial populations lining the airways that are
10:29:31 7 referred to as "neuroendocrine cells" that show changes
10:29:38 8 that are associated with functional changes in the
10:29:43 9 respiratory system and that, again, critical windows of
10:29:50 10 exposure during perinatal development with exposure to
10:29:57 11 environmental tobacco smoke can also lead to changes in
10:30:02 12 pulmonary function that will persist after exposure to
10:30:07 13 environmental tobacco smoke ends.
10:30:18 14 Q. Is there anything else?
10:30:22 15 A. Those will be the major topics.
10:30:32 16 Q. Okay. As we -- as you walk through that
10:30:35 17 list that you just described for me, are you going to
10:30:39 18 be talking about these -- you talked about a lot of
10:30:41 19 changes and cellular changes. Are you going to be
10:30:44 20 talking about those changes in animals, humans or both?
10:30:48 21 A. Primarily in animals, but we'll confirm it
10:30:52 22 with studies that have been done in humans.
10:30:57 23 Q. Your studies have been limited to looking at
10:30:59 24 those issues in animals, correct?
10:31:01 25 A. That is correct.
10:31:05 26 Q. And when we talk about animals, actually, am
10:31:08 27 I correct in saying that your studies have been limited
10:31:10 28 to looking specifically at rats?

10:31:16 1 A. No.
10:31:16 2 Q. What other animals?
10:31:16 3 A. Mice.

10:31:18 4 Q. Any other animals?
10:31:19 5 A. Guinea pigs. Rhesus monkeys.
10:31:47 6 Q. Okay. As -- I tried to write down as fast
10:31:49 7 as I could... when we talked about prenatal effects, I
10:31:52 8 believe you mentioned two -- the small reduction in
10:31:57 9 fetal body weight and the change in the maturation of
10:32:01 10 the epithelial cells?
10:32:03 11 A. Lining the airways.
10:32:04 12 Q. Lining the airways? Did I miss any?
10:32:07 13 A. No. That's correct.
10:32:10 14 Q. Could you describe for me... What is the
10:32:15 15 significance of a change in the maturation of
10:32:19 16 epithelial cells lining the airways?
10:32:24 17 A. Well, there is very critical points in which
10:32:28 18 epithelial cells must undergo differentiation and
10:32:33 19 maturation, and so for those types of changes to occur
10:32:41 20 during fetal development, we don't know what their
10:32:47 21 potential long-term sequelae may be, but it is
10:32:51 22 certainly a deviation from the normal developmental
10:32:55 23 process.
10:32:57 24 Q. Are there any disease end points that that's
10:33:00 25 known to cause?
10:33:05 26 A. Maturation of epithelial cells in a
10:33:09 27 different pattern, yes. These have been associated
10:33:16 28 with conditions of asthma. They've been associated

10:33:22 1 with conditions of bronchopulmonary dysplasia as the
10:33:30 2 two primary things that I can think of.
10:33:34 3 Q. Have you actually studied as to whether in
10:33:37 4 your experiments those lead -- those changes lead to
10:33:40 5 asthma?
10:33:43 6 A. Only from the perspective that we've looked
10:33:45 7 at, pulmonary function in the animals postnatally, to
10:33:53 8 test whether there are changes in lung resistance or in
10:34:00 9 airway compliance.
10:34:03 10 Q. And what have you found there?
10:34:04 11 A. That there are significant alterations if
10:34:10 12 exposure to environmental tobacco smoke occurs both
10:34:13 13 in utero as well as postnatally.
10:34:19 14 Q. What if exposure to ETS only occurs
10:34:22 15 in utero?
10:34:24 16 A. Then we see -- we know that we see the
10:34:27 17 epithelial changes, but postnatally, we do not see any
10:34:32 18 changes in pulmonary function.
10:34:35 19 Q. And what if the ETS exposure occurs only
10:34:39 20 postnatally?
10:34:41 21 A. Again, we see changes in epithelial
10:34:44 22 maturation and expression of biochemical components,
10:34:50 23 but we do not see changes in lung function.
10:34:56 24 Q. So when you see the changes in the -- I need
10:34:59 25 you to say that one for me again. You saw changes
10:35:01 26 where?
10:35:02 27 A. Postnatally?
10:35:03 28 Q. Yes.

10:35:04 1 A. We saw them occurring within the same cell
10:35:08 2 types, that we saw changes in the in utero. These are
10:35:14 3 epithelial cells lining the airways.
10:35:18 4 Q. Okay. So when you saw exposure only
10:35:21 5 postnatally and you saw changes in the epithelial
10:35:24 6 lining the airways but no changes in lung function,

what was the significance of the changes in the epithelial lining of the airways?

A. That there was a persistent up regulation of metabolic function that was not present in the age match littermate controls, again, done in rats.

Q. In rats?

A. Uh-huh.

Q. Okay. Are there any other changes -- we talked about those two, and we talked about the postnatal. Are there any other effects on lung growth and development that you intend to testify about?

A. Well, no.

Q. And I think you answered this before, but let me just make sure.

You intend to only testify about changes in lung growth and development. Are there any other areas that we haven't covered that you intend to testify about?

A. No.

MR. KODSI: Okay. Actually, we've gone about an hour and we've run through some things, so why don't we take about five, if that's okay, or ten.

THE VIDEOGRAPHER: Going off the record, the time is 10:36.

(Recess taken)

(Mr. Cafferty and Ms. Moore are not present.)

THE VIDEOGRAPHER: Back on the record, the time is 10:49.

MR. KODSI: Q. Okay. Just to kind of sum up the points we were just making, Dr. Pinkerton, I may need your help if you don't understand the way I'm phrasing this, but we've talked about a lot of cellular changes. Are there any particular disease end points that you intend to testify that ETS causes?

A. Yes, increased airway reactivity.

Q. Are there any others?

A. There are changes in metabolic function, but I would not conclude from our studies that we can define those as a "disease end point."

Q. Is there a different term I should use other than "disease end point"? Well, let's start with increased airways reactivity first.

A. Uh-huh.

Q. Is it your opinion that ETS causes increased airways reactivity in humans?

A. Yes.

Q. And that is -- when I said, "humans," do you believe it causes increased airway reactivity in both children and adults?

A. More so in children.

Q. And what is your basis for that opinion?

A. The prevalence of asthma in children who live in homes where smoking occurs.

Q. So that is the epidemiologic literature on ETS and childhood asthma?

A. Yes.

Q. Have you reviewed all of the available epidemiologic studies on ETS and childhood asthma?

10:51:26 10 A. I've reviewed quite a few.
10:51:29 11 Q. About how many?
10:51:33 12 A. Well, most of the reviews that I've really
10:51:40 13 been involved with have taken place since publications
10:51:45 14 of probably the early '70s to date, so I would think
10:51:51 15 that those would include a minimum 30 to 40 different
10:51:58 16 studies.
10:52:01 17 Q. And you are aware there are some
10:52:04 18 epidemiologic studies that have looked at ETS and
10:52:08 19 childhood asthma and concluded that they could not find
10:52:11 20 an association?
10:52:11 21 MR. BROOKEY: Objection; lacks foundation.
10:52:12 22 He can answer.
10:52:15 23 THE WITNESS: I'm aware that there are some
10:52:16 24 studies that show that -- that do not show the same
10:52:23 25 effects as seen in the majority of other studies.
10:52:28 26 MR. KODSI: Q. So that that literature on
10:52:29 27 ETS and childhood asthma is inconsistent? Would that
10:52:32 28 be a fair way to characterize it?

10:52:37 1 A. I would think that due to different exposure
10:52:40 2 conditions, different environments in terms of the
10:52:45 3 background of the children, the parameters that were
10:52:49 4 being measured that that also could account for
10:52:55 5 different results from different epidemiological
10:53:00 6 studies.
10:53:02 7 Q. And you're also relying, I assume, on animal
10:53:05 8 studies for your opinion that ETS causes increased
10:53:07 9 airways reactivity?
10:53:09 10 A. Yes.
10:53:11 11 Q. Are you relying on any studies other than
10:53:13 12 those conducted by yourself?
10:53:16 13 A. Those are the primary studies.
10:53:24 14 Q. What other -- what animal studies other than
10:53:26 15 your own have you reviewed in that area?
10:53:33 16 A. Well, there have been a number of studies
10:53:35 17 that have looked at cigarette smoke and lung
10:53:40 18 development.
10:53:42 19 In terms of lung function, I don't recall
10:53:48 20 the exact studies that have been done, but they've been
10:53:52 21 rather limited, and therefore, that is the reason why
10:53:55 22 I'm putting more emphasis on our own studies that we've
10:54:00 23 done recently than trying to look at other animal
10:54:05 24 studies. I think the proof of airway reactivity
10:54:11 25 associated with environmental tobacco smoke is based in
10:54:15 26 humans more so than in a lot of animal studies.
10:54:34 27 Q. Do you intend to testify about then the
10:54:37 28 human epidemiology on airways reactivity?

10:54:42 1 A. Only as it relates to our own studies in
10:54:46 2 animal models, particularly with the critical windows
10:54:51 3 of exposure that I've explained.
10:55:02 4 Q. Okay. Now, you said it's your opinion that
10:55:08 5 ETS exposure causes increased airways reactivity.
10:55:11 6 Is there anything else that ETS exposure
10:55:13 7 causes that you intend to testify about?
10:55:21 8 A. No.
10:55:23 9 Q. Now, is increased airways reactivity the
10:55:25 10 same as asthma?
10:55:29 11 A. They have similar interpretations. Asthma
10:55:34 12 is associated with increased airway reactivity.

10:55:37 13 Q. Do you intend to testify that ETS exposure
10:55:40 14 causes asthma?
10:55:43 15 A. Only from the perspective of the
10:55:44 16 epidemiological studies. Our animal studies show
10:55:53 17 similar effects, but I think it would be inappropriate
10:55:59 18 to say that we create asthma in rats, but we do create
10:56:07 19 increased airway reactivity, which is a hallmark of
10:56:13 20 asthma.
10:56:16 21 Q. Now, earlier, we talked about your
10:56:20 22 description of the epidemiology in this case will
10:56:23 23 relate to the animal studies that you've conducted.
10:56:27 24 Is that a fair characterization?
10:56:28 25 A. Yes.
10:56:30 26 Q. So do you intend to testify about the ETS
10:56:33 27 epidemiology on ETS exposure in asthma?
10:56:39 28 A. Again, based on the relevant epidemiological

10:56:43 1 studies that show that there is an association of
10:56:47 2 environmental tobacco exposure and increased incidence
10:56:52 3 of asthma and what the functional consequences of those
10:56:55 4 things are as it relates -- yes, I would testify on
10:56:59 5 that, because it has direct relevance to our own
10:57:03 6 experimental laboratory studies.
10:57:17 7 MR. KODSI: Okay. Let me mark -- and this
10:57:20 8 is the declaration for this. Brian, this is just the
10:57:41 9 expert declaration.
10 (Whereupon, Defendants' Exhibit 528 was
11 marked for identification.)
10:57:42 12 MR. KODSI: Q. Dr. Pinkerton, we've handed
10:57:44 13 you what the court reporter has just marked as
10:57:46 14 Exhibit 528, and let me ask you if you recognize that
10:57:50 15 document.
10:57:51 16 A. Yes.
10:57:52 17 Q. Okay. And that is the declaration that you
10:57:54 18 filed in this case in, I believe, October of '99?
10:58:06 19 A. Yes.
10:58:13 20 Q. Could you describe for me briefly how you
10:58:15 21 prepared that declaration?
10:58:23 22 A. This involved a general literature review
10:58:30 23 with the focus being on environmental tobacco smoke,
10:58:37 24 lung development, with an emphasis both on the
10:58:41 25 epidemiology of this in children as well as in any
10:58:45 26 studies where animals may have been involved with
10:58:51 27 exposures to cigarette smoke.
10:58:56 28 It included also providing information about

10:59:00 1 my background as a scientist and as a faculty member at
10:59:07 2 the University of California and my research interests
10:59:12 3 with the particular emphasis on environmental tobacco
10:59:16 4 smoke.
10:59:17 5 It also includes a summary of our
10:59:24 6 experiments and our research on environmental tobacco
10:59:29 7 smoke as completed in animal -- laboratory animals over
10:59:35 8 the last eight to nine years performed at the
10:59:41 9 University of California.
10:59:52 10 It also involved, basically, the writing of
10:59:52 11 a number of drafts and rewriting and then a completion
10:59:59 12 of everything, with references to document what is
11:00:05 13 found in the declaration.
11:00:09 14 Finally, there is actually two parts to this
11:00:13 15 declaration. One, the second part talks about the

11:00:18 16 respiratory system and critical windows of exposure for
11:00:22 17 children's health.
11:00:25 18 Q. And, just for the record so we know, that
11:00:26 19 part begins on Page 16 --
20 A. Yes.
11:00:28 21 Q. -- of Exhibit 528? Okay. It was just the
11:00:31 22 page you were looking at, just to clarify.
11:00:34 23 A. This was prepared not originally as part of
11:00:38 24 the declaration but simply as a further explanation for
11:00:46 25 what is involved in the process of perinatal lung
11:00:53 26 development and how environmental factors may impact on
11:00:58 27 lung development, cellular differentiation, branching
11:01:05 28 morphogenesis, which included, as just one example, the

11:01:11 1 effects of exposure to environmental tobacco smoke
11:01:15 2 during critical windows of perinatal development, and
11:01:25 3 that was actually included as part of the declaration.
11:01:29 4 It was originally not meant to be so but was one that
11:01:38 5 contributed to an overall understanding of what is
11:01:43 6 involved during perinatal development and potential
11:01:47 7 effects of environmental tobacco smoke on that period
11:01:51 8 of development.
11:01:51 9 (Ms. Moore rejoins the proceedings.)
11:01:53 10 MR. KODSI: Q. When you began preparing
11:01:55 11 Exhibit 528 -- and we'll just call Exhibit 528 "the
11:01:59 12 declaration" for -- or...
11:02:01 13 When you began preparing the declaration,
11:02:04 14 what did you understand the goal of it to be? What was
11:02:08 15 the purpose of it?
11:02:09 16 A. The purpose, as I understand it, was to
11:02:13 17 discuss health effects associated with exposure to
11:02:20 18 environmental tobacco smoke during development of the
11:02:25 19 respiratory system in the perinatal period with an
11:02:32 20 emphasis on animal models and how those studies relate
11:02:39 21 to human studies, human epidemiological studies of
11:02:47 22 children's health effects with exposure to
11:02:49 23 environmental tobacco smoke.
11:02:51 24 Q. And I should have done this before.
11:02:53 25 We've talked about the definitions of
11:02:55 26 "prenatal" and "postnatal," and I have not yet asked
11:02:58 27 you to define "perinatal." So for clarification, if
11:03:01 28 you could define that for the record...

11:03:03 1 A. "Perinatal" refers to both the pre and
11:03:06 2 postnatal periods of lung development.
11:03:10 3 Q. And the perinatal period would end as an
11:03:14 4 animal approaches adulthood as well?
11:03:17 5 A. That is correct.
11:03:21 6 Q. Okay. Now, did you receive any assistance
11:03:23 7 in preparing your declaration, Exhibit 528?
11:03:28 8 A. Only from the perspective of my background.
11:03:36 9 I provided my CV to the firm Preston,
11:03:45 10 Gates & Ellis, and from that CV, they prepared parts of
11:03:49 11 my introduction -- where I went to school, what my
11:03:55 12 background was, some of the research funding that I had
11:04:02 13 received relevant to studies of environmental tobacco
11:04:05 14 smoke.
11:04:09 15 Q. Now, in discussing your background, were
11:04:11 16 there any areas that they included that aren't in the
11:04:16 17 draft we see now that you were uncomfortable with and
11:04:18 18 asked to be taken out?

11:04:20 19 A. No.
11:04:21 20 Q. Were there any areas that they left out that
11:04:23 21 you felt you needed to have included?
11:04:26 22 A. Not for this particular topic.
11:04:28 23 Q. All right. Did you receive any assistance
11:04:30 24 in drafting the other portions of your declaration?
11:04:33 25 A. No.
11:04:34 26 Q. So you drafted that by yourself?
11:04:37 27 A. Yes.
11:04:39 28 Q. All right. Now -- and I guess when I'm

11:04:41 1 saying, "any assistance," I'm not just limiting it to
11:04:43 2 attorneys. People in your lab? Dr. Joad?
11:04:46 3 No assistance from anyone else?
11:04:48 4 A. That's correct.
11:04:48 5 Q. Okay. I just wanted to make sure that my
11:04:50 6 question was clear.
11:04:57 7 You talked about the drafting process of the
11:04:59 8 declaration. Did you send preliminary drafts to anyone
11:05:03 9 for their review?
11:05:05 10 A. No.
11:05:05 11 Q. So the drafting process was all you just
11:05:07 12 revising your own drafts?
11:05:10 13 A. That's correct.
11:05:19 14 Q. Okay.
11:05:19 15 A. Perhaps --
11:05:19 16 Q. Okay.
11:05:20 17 A. -- with -- with Carolyn Sieve at Preston,
11:05:28 18 Gates & Ellis. I sent her a draft of the declaration
11:05:36 19 and the following week -- I went to a workshop, and the
11:05:39 20 following week, I might have sent her a second draft
11:05:43 21 with minor corrections.
11:05:47 22 Q. But did Ms. Sieve ever suggest any
11:05:49 23 corrections that you needed?
11:05:50 24 A. No.
11:06:00 25 Q. Okay. Let's start by going through the
11:06:02 26 declaration in Paragraph 10. If you could flip to
11:06:07 27 that, it begins on Page 3.
11:06:25 28 If you look there, in Paragraph 10, you

11:06:31 1 state that -- I'm looking at Line 20 -- it is clear
11:06:37 2 that active smoking is associated with a variety of
11:06:40 3 respiratory diseases. Then you give a list, and then
11:06:47 4 on Line 23, you state that growing evidence suggests
11:06:52 5 that many of these same disease processes can also
11:06:56 6 occur through passive exposure to cigarette smoke.
11:07:00 7 Do you see what I'm talking about there?
11:07:02 8 A. Yes.
11:07:03 9 Q. Could you just explain for me why you chose
11:07:06 10 the phrase "it is clear" when you were talking about
11:07:09 11 active smoking, but yet you didn't say, "it is clear,"
11:07:14 12 for passive smoking?
11:07:19 13 A. I think that the effects of active smoking
11:07:22 14 based on the fact that a much greater dose of cigarette
11:07:29 15 smoke is taken into the respiratory system leads to
11:07:35 16 very clear-cut effects, health effects.
11:07:40 17 Passive exposure to tobacco smoke is
11:07:46 18 delivered to the respiratory system at a much lower
11:07:49 19 dose and in a much more variable condition, and
11:07:55 20 therefore, those health effects that are associated
11:08:05 21 with it were not recognized as soon or as clearly as

active smoking health effects were recognized.
Q. When you say, "were recognized," you're talking about in the past, I assume, but --
A. Uh-huh.
Q. -- at the time you wrote your declaration, you still chose not to use the phrase "it is clear" for passive smoking. Why didn't you choose to use that in

the current time?
A. Uh-huh. I think, as a scientist, we always want to be very careful about making absolute statements. Although I think that the epidemiology provides some very solid evidence that there are similar health effects associated with passive exposure to cigarette smoke that are seen in active smokers, that -- it's just something about us as scientists that we always want to hold back and not make too many definitive statements, you know, such as that.
So I guess that's my explanation for the use of that terminology.
Q. What was it that made you more comfortable about the active smoking data that allowed you to use the phrase "it is clear" when you were talking about active smoking?
A. I think it's based on the literature and the scientific evidence that is available, and that is really undeniable, that there are health effects associated with active smoking that include those things that were stated, such as the cardiovascular disease in addition to respiratory diseases.
Q. Is it then safe to say that -- and I hate to use a double negative -- but that the evidence, the epidemiologic evidence on ETS, is not undeniable?
MR. BROOKEY: Objection. That's vague and ambiguous and misstates prior testimony, but he can answer it if he understands.

THE WITNESS: Would you restate it?
MR. KODSI: Yes.
Q. You said that the evidence on active smoking is undeniable. How do you define -- when you said that, I want to ask you to define -- maybe we can do this better. Define what you mean by "undeniable."
A. A cause and effect association is present.
Q. And when you say, "undeniable," do you mean that it's really not debated in the scientific community anymore? Is that maybe what you mean by "undeniable"?
A. For active smoking?
Q. For active.
A. Yes.
Q. Would you agree that with respect to ETS exposure that there still is debate in the scientific community as to whether or not it causes the diseases you've listed here?
A. No. I don't think that there is any -- any debate about it any longer.
Q. Okay. For which diseases do you think that there is no debate?
A. An increase of respiratory infections, an increase of asthma in children, an increase in the

11:11:33 25 severity of the type of asthmatic attacks that children
11:11:37 26 have associated with exposure to environmental tobacco
11:11:42 27 smoke, and also reductions in growth with maternal
11:11:56 28 exposure to environmental tobacco smoke during

11:11:58 1 pregnancy of the child, itself, and I guess included
11:12:04 2 in -- rather than just saying that it's a broad
11:12:08 3 statement that asthma and environmental tobacco smoke
11:12:11 4 show these associations, I think it would be more fair
11:12:14 5 to say that there are decrements in pulmonary function
11:12:20 6 that are clearly associated with passive exposure to
11:12:25 7 environmental tobacco smoke in children that are
11:12:30 8 influenced by exposure during early childhood, and that
11:12:36 9 may be actually exacerbated by exposure during
11:12:41 10 pregnancy for that child, and again, I'm keeping my
11:12:47 11 comments confined to children.
11:12:51 12 Q. And, in that answer, were you also keeping
11:12:54 13 your comments confined to the diseases or health
11:13:00 14 effects that you intend to testify at trial?
11:13:05 15 A. For the most part, I would say, yes, because
11:13:07 16 of the fact that it's really the children that I'm...
11:13:13 17 and our model really best represents effects that may
11:13:18 18 be manifested in children rather than in adults.
11:13:24 19 Q. Okay. So -- and I think my first question
11:13:27 20 was whether there was no longer any debate in the
11:13:30 21 scientific community for certain health effects that
11:13:33 22 ETS might cause, and I want to make -- I'll just read
11:13:35 23 back what I've got and make sure that I've got this
11:13:38 24 right.
11:13:38 25 So is it your opinion that there is no
11:13:40 26 longer any debate in the scientific community as to
11:13:43 27 whether or not ETS causes increased respiratory
11:13:47 28 infection?

11:13:49 1 A. In children?
11:13:49 2 Q. In children.
11:13:51 3 A. That's correct.
11:13:54 4 Q. Okay. And is it your opinion that there is
11:13:57 5 no longer any debate in the scientific community as to
11:14:00 6 whether or not ETS exposure causes increased asthma in
11:14:05 7 children?
11:14:05 8 A. Yes. That's correct.
11:14:07 9 Q. And it's your opinion that there is no
11:14:09 10 longer any debate in the scientific community as to
11:14:12 11 whether ETS exposure causes increased severity of
11:14:16 12 asthma?
11:14:24 13 A. I would -- I would have to say that I'm not
11:14:26 14 sure --
11:14:28 15 Q. Okay.
11:14:29 16 A. -- about that.
11:14:29 17 Q. So you think there may be debate in the
11:14:31 18 scientific community as to whether or not ETS causes
11:14:34 19 increased severity of asthma in children?
11:14:41 20 A. I would say that more likely than not that
11:14:44 21 it does cause an increased severity in the type of
11:14:48 22 asthmatic attacks that children have, but I don't have
11:14:55 23 at my disposal right at the moment to say that I can
11:14:59 24 quote a paper of that nature.
11:15:09 25 Q. When you say you don't have it at the moment
11:15:13 26 at your disposal, the paper, are you aware of any such
11:15:18 27 paper or -- that you didn't bring with you or is it

your -- is that really outside the scope of what your

knowledge base is?

A. No. I think it's just that I don't have that information with me, but I know that through the review of the epidemiological literature that that is what is reported, that not only is there an increase in the incidence of asthma, but there is also an increase in the severity of asthmatic attacks and actually the frequency of asthmatic attacks among children who are exposed to secondhand cigarette smoke.

Q. And are you aware of epidemiologic studies that report the opposite of that?

A. No.

Q. And the next one I have written down is it is your opinion that there is no longer any debate in the scientific community as to whether or not ETS causes decrease in a growth reduction with respect to maternal exposure.

A. That is correct.

Q. Okay. And the last one I wrote down is it's your opinion that there is no longer any debate in the scientific community as to whether or not ETS exposure causes decrements in pulmonary function.

A. In children.

Q. In children.

A. Uh-huh.

Q. Thank you for the correction.
Are there any that I missed?

A. Well, and I think that an important part of

this to keep in mind is that these decrements in pulmonary function that are seen in children -- we're now beginning, I think through the epidemiological literature and studies that are going on, to suggest that these are actually persistent decrements. They're not something that goes away with further development in aging of these children into adolescence, that these decrements are still present.

Q. And you're relying on the epidemiologic data for that, correct?

A. Yes.

Q. Do you have any animal data that supports that?

A. Yes. Basically, we have done studies which -- where we have stopped exposures to environmental tobacco smoke at that point where the animals have -- or almost have reached sexual maturity; they're not quite adults, but we just take them out totally of any exposures and we maintain them in filtered air, and we've been able to show that they have persistent increased airway reactivity even after they've been out of -- out of smoke exposures for an extended period of time.

Q. Okay. Now I'd like to actually ask you a question going back to the declaration, Paragraph 58, which is on Page 26, and I wanted to focus on the last sentence there.

It says, "Our knowledge base regarding

perinatal exposure and critical windows is negligible. Future studies must be designed to address these central issues to better understand and provide meaningful data to benefit the health of children during development as well as into adulthood of these individuals." I've read that correctly?

A. (Nods head.)

Q. Could you explain for me what you meant there?

A. This second part of the declaration, as I mentioned earlier, was something that was not originally prepared for this declaration but for a workshop held by the US Environmental Protection Agency last fall in Richmond, Virginia, and my assignment at that time was to discuss critical windows of exposure for children's health with regard to the respiratory system.

So the point was not to focus on just environmental tobacco smoke but environmental factors in general, and one glaring gap that really became obvious to me is the fact that perinatal development, although we are getting a better handle on understanding just exactly the steps that are important in that process to occur and that it's a multi-step process, that it doesn't end with the birth of a child but actually continues postnatally, is very critical in better understanding how exposure to environmental pollutants may be different in a child than it is in an

adult.

And so that's what I meant by that statement is that we don't have a very good database with regard to environmental air pollutants, in general, as to just what are the critical windows for exposure to an environmental pollutant that may affect the development of the respiratory system of that child and may have lasting effects into adulthood. So does that clarify?

(Ms. Moore leaves the room and Mr. Cafferty rejoins the proceedings.)

MR. KODSI: I think so.

Q. Let me ask: You would agree then that the knowledge base regarding perinatal exposure to ETS and critical windows is also negligible?

A. Not to the same extent with other environmental pollutants, and again, that is the basis of my testimony is that, through our studies where we've actually been able to use very controlled, experimental conditions where we characterize our exposure conditions to cigarette smoke and then also control which windows are -- during development that exposures occur, whether it be a maternal exposure so it's an in utero exposure to the fetus or it's a postnatal exposure, either alone or in combination allows us to get a better handle on what are critical windows of exposure, at least for environmental tobacco smoke that we create in our laboratory, in our research.

Q. Now, you said it's not as negligible as for other air pollutants.

A. Right.

11:22:13 4 Q. Do you feel that -- in your statement that
11:22:14 5 future studies need to be designed, do you agree that
11:22:17 6 future studies still need to be done on ETS?
11:22:20 7 A. I think it's still an ongoing process.
11:22:26 8 Q. And why is it still an ongoing process?
11:22:30 9 A. Although we've been able to document the
11:22:35 10 health effects that are seen in humans in our animal
11:22:39 11 studies, we still don't have the mechanisms by which
11:22:46 12 these changes may be occurring. We know that they're
11:22:50 13 reproducible, that they are associated with exposure to
11:22:55 14 environmental tobacco smoke and during these periods of
11:22:59 15 perinatal development, but we still need to understand
11:23:04 16 exactly how these changes come about. Even though
11:23:08 17 they're highly reproducible, study after study that we
11:23:13 18 do, we don't have the cellular or the molecular basis
11:23:18 19 by which these effects are occurring.
11:23:21 20 Q. And one of the purposes for the animal
11:23:24 21 studies that you're conducting is to try to determine,
11:23:28 22 as you said, the mechanism for these effects.
11:23:30 23 A. Uh-huh.
11:23:32 24 Q. Is that correct?
11:23:32 25 A. That's correct.
11:23:33 26 Q. And currently, in your opinion, we don't
11:23:35 27 understand the mechanism for an increased respiratory
11:23:40 28 infection in children?

11:23:43 1 A. Well, I think there is evidence for what may
11:23:47 2 be associated with that, that there can be changes in
11:23:53 3 the immune system of the respiratory system that may
11:23:57 4 lead to increased respiratory infection, that there may
11:24:01 5 also be changes in the way that things are cleared from
11:24:07 6 the lungs through the airways that may also influence
11:24:13 7 respiratory infections and the clearance of bacterial
11:24:19 8 or viral agents that may be inhaled. So I think that
11:24:23 9 there are some leads that we have to explain exactly
11:24:31 10 how that may be occurring.
11:24:32 11 Q. But still more work needs to be done, in
11:24:35 12 your opinion?
11:24:36 13 A. I think so.
11:24:37 14 Q. Okay. And, as you said, what I'd like to do
11:24:40 15 is ask these questions walking through the diseases
11:24:42 16 that we just talked about.
11:24:44 17 It's your opinion that we still don't
11:24:46 18 understand the mechanisms for increased asthma in
11:24:49 19 children?
11:24:54 20 A. Based on exposure to environmental tobacco
11:24:56 21 smoke?
11:24:57 22 Q. Good clarification. Yes, based on exposure
11:25:00 23 to environmental tobacco smoke.
11:25:02 24 A. I think that's correct.
11:25:04 25 Q. And it's your opinion that we still don't
11:25:07 26 understand the mechanisms that might cause increased
11:25:12 27 severity of asthma based on exposure to environmental
11:25:15 28 tobacco smoke?

11:25:17 1 A. That is correct.
11:25:17 2 Q. And that we still don't understand the
11:25:19 3 mechanisms that might be responsible for decreased
11:25:25 4 growth based on maternal exposure to environmental
11:25:28 5 tobacco smoke?
11:25:32 6 A. That's correct.

11:25:32 7 Q. And that we don't understand the mechanisms
11:25:35 8 that are responsible for decrements in pulmonary
11:25:41 9 function in children based on exposure to environmental
11:25:44 10 tobacco smoke?
11:25:46 11 A. Well, from some of our studies, I think we
11:25:49 12 can attribute some of these changes based on just an
11:25:55 13 alteration in the growth of the respiratory system.
11:26:00 14 There's also studies to suggest that there may be
11:26:03 15 changes in some of the cellular compartments, smooth
11:26:09 16 muscle within the airways.
11:26:12 17 Q. Was that the bronchial smooth muscle?
11:26:14 18 A. Uh-huh, or changes in the epithelial lining
11:26:21 19 of the airways that may influence the amount of
11:26:25 20 secretion of certain fluids or -- that may also affect
11:26:32 21 airway tone. So it's not as though we don't know --
11:26:37 22 don't have any idea about the mechanisms. There are
11:26:40 23 certainly very, very clear-cut examples of things that
11:26:45 24 may be occurring that might explain these decrements in
11:26:50 25 pulmonary function.
11:26:51 26 (Ms. Moore rejoins the proceedings.)
11:26:52 27 MR. KODSI: Q. But still more work needs to
11:26:54 28 be done to understand the mechanisms?

11:26:56 1 A. That's correct.
11:26:56 2 Q. And I've probably used a poor word choice.
11:26:59 3 I said, "we don't understand," when we were talking
11:27:01 4 about the mechanisms, but it's really science --
11:27:04 5 scientists. I don't want to lump myself in that group.
11:27:07 6 I'm not there yet.
11:27:08 7 Scientists don't understand the mechanisms
11:27:10 8 that we've just talked about, correct?
11:27:12 9 A. To the degree that they would like to know
11:27:15 10 them --
11:27:15 11 Q. Right.
11:27:16 12 A. -- yes.
11:27:20 13 Q. Okay. We've talked about each of these
11:27:22 14 disease ends points or health effects.
11:27:24 15 When did you first reach the conclusion that
11:27:25 16 ETS causes increased respiratory infection in children?
11:27:34 17 A. That was based on my initial review of the
11:27:36 18 literature, and so my conclusion was based on what
11:27:44 19 other studies had found in terms of looking at children
11:27:47 20 and respiratory infection.
11:27:50 21 Q. Is that a review of the literature you
11:27:52 22 conducted specifically for this case or is that a
11:27:54 23 review of the literature you conducted in the past?
11:27:57 24 A. It's a review of the literature that I
11:27:59 25 conducted in the past for the purposes of understanding
11:28:02 26 health effects of environmental tobacco smoke.
11:28:05 27 Q. And do you have a time frame as to when you
11:28:07 28 conducted that review and reached the conclusion to

11:28:16 1 your satisfaction that ETS causes increased respiratory
11:28:16 2 infection in children?
11:28:16 3 A. I would say that that was probably a
11:28:18 4 conclusion that I reached in 1993 with the introduction
11:28:27 5 of an EPA document that reviewed extensively the
11:28:34 6 literature on health effects of passive exposure to
11:28:38 7 cigarette smoke in the human population.
11:28:42 8 Q. Would that be the EPA's 1992 report on
11:28:45 9 environmental tobacco smoke?

11:28:46 10 A. That's correct.
11:28:46 11 Q. Was that the primary basis for your opinion
11:28:49 12 that ETS causes increased respiratory infection in
11:28:53 13 children?
11:28:56 14 A. That was a very good resource for me to be
11:29:01 15 able to start looking into that issue, and so I
11:29:08 16 wouldn't say that it's based on that report but more on
11:29:11 17 the fact that there -- that that was a compilation of a
11:29:15 18 number of epidemiological studies which looked into all
11:29:19 19 sorts of health effects, including increased
11:29:23 20 respiratory infection.
11:29:25 21 Q. Now, have you reviewed the original
11:29:29 22 epidemiologic studies on ETS and increased respiratory
11:29:34 23 infection or did you review reviews of those studies?
11:29:38 24 A. I have not -- I have not reviewed every
11:29:42 25 epidemiological study on increased respiratory
11:29:47 26 infection in children with ETS exposure.
11:29:50 27 Q. You've reviewed some of them?
11:29:52 28 A. Some of them, yes.

11:29:53 1 Q. About what -- do you have in mind the
11:29:55 2 percentage you've reviewed? Again, we're talking about
11:29:59 3 the actual studies themselves, not --
11:30:01 4 A. Uh-huh.
11:30:02 5 Q. -- abstracts or...
11:30:03 6 A. Uh-huh. Uh-huh. The number of
11:30:08 7 epidemiological studies that would include also looking
11:30:11 8 at pulmonary infection as an end point would probably
11:30:15 9 be on the order of 10 to 15 of the original studies.
11:30:24 10 Again, these would be from the peer-reviewed literature
11:30:30 11 in reputable scientific journals.
11:30:38 12 Q. Anything other than epidemiologic studies
11:30:41 13 that led you to the conclusion that ETS causes
11:30:44 14 increased respiratory infection in children?
11:30:47 15 A. No.
11:30:52 16 Q. Now, you had indicated when we listed these
11:30:55 17 diseases the first time that you don't think that there
11:30:57 18 is any debate in the scientific community that ETS
11:31:02 19 causes increased respiratory infection in children,
11:31:04 20 correct?
11:31:06 21 A. That's correct.
11:31:06 22 Q. Are you aware of any individual scientists
11:31:09 23 that disagree with that conclusion?
11:31:13 24 A. No.
11:31:19 25 Q. Let's go to the next one.
11:31:21 26 When did you first reach the conclusion that
11:31:23 27 ETS causes increased asthma in children?
11:31:30 28 A. Again, this would be through our initial

11:31:34 1 review of the literature. We first started doing
11:31:38 2 studies with environmental tobacco smoke, studies in
11:31:41 3 probably 1990/1991 through funding that we had received
11:31:47 4 to specifically look at ETS and lung growth in
11:31:52 5 children, and so I would say that it was probably
11:31:55 6 around that time that we first started noticing in the
11:32:00 7 literature that there was an association between asthma
11:32:03 8 in children and ETS, but I think, again, the document
11:32:09 9 that provided me with the greatest compilation of
11:32:14 10 everything had to be the EPA 1992 document.
11:32:18 11 Q. So it would be after reviewing the EPA 1992
11:32:21 12 document that you first were comfortable with the

11:32:24 13 conclusion that ETS causes increased asthma in
11:32:27 14 children?
11:32:29 15 A. Only from the perspective that it provided a
11:32:32 16 more comprehensive review of the literature with --
11:32:37 17 regarding children's health and asthma and ETS
11:32:40 18 exposures.
11:32:47 19 Q. Now, you had indicated that the reason you
11:32:52 20 had started reviewing some of this literature back in
11:32:54 21 1991 was funding you had received to study ETS?
11:32:59 22 A. Yes.
11:32:59 23 Q. What was the source of that funding?
11:33:02 24 A. It came from the Center for Indoor Air
11:33:11 25 Research.
11:33:11 26 Q. Okay. We'll probably probe a little bit of
11:33:13 27 that later, but I just wanted to go ahead and clarify
11:33:17 28 that --

11:33:18 1 A. Okay.
11:33:19 2 Q. -- while you mentioned it.
11:33:20 3 Now, what is the basis or what was the basis
11:33:23 4 for -- scratch all that.
11:33:24 5 You've indicated you reviewed epidemiologic
11:33:32 6 studies that you relied upon for reaching the
11:33:35 7 conclusion that ETS causes increased asthma in
11:33:39 8 children, and you reached that conclusion by 1993,
11:33:45 9 correct?
11:33:46 10 A. Correct.
11:33:46 11 Q. That's the head nod issue we talked about
11:33:49 12 earlier. Were there any studies other than
11:33:52 13 epidemiologic studies that led you to that conclusion
11:33:57 14 in 1993?
11:33:59 15 A. No.
11:34:04 16 Q. Okay. Let's talk about the next one.
11:34:06 17 When did you first reach the conclusion that
11:34:10 18 ETS exposure causes increased severity of asthma in
11:34:18 19 children?
11:34:20 20 A. Again, this would be through the review of
11:34:22 21 the literature during the same period of time, 1991
11:34:27 22 through 1993, and the increased severity of asthma
11:34:34 23 really was brought to my attention through the 1992 EPA
11:34:38 24 document.
11:34:46 25 Q. And, again, the review of the literature
11:34:48 26 that led you to this conclusion was a review that you
11:34:51 27 did under funding from the Center for Indoor Air
11:34:56 28 Research?

11:34:56 1 A. That is correct.
11:34:57 2 Q. And if we refer to that -- I think you did
11:34:59 3 already, but if we refer to that as "CIAR" from now on,
11:35:06 4 you'll understand what I'm talking about?
11:35:06 5 A. That's --
6 Q. Okay.
11:35:06 7 A. That would be fine.
11:35:07 8 Q. I know the court reporter would prefer that
11:35:08 9 any chance we get.
11:35:10 10 And your opinion that ETS causes an increase
11:35:16 11 in the severity of asthma, is that based on anything
11:35:20 12 other than epidemiologic studies?
11:35:23 13 A. No.
11:35:28 14 Q. Okay. Now let's go to your opinion that ETS
11:35:32 15 causes a decrease in growth with maternal exposure to

ETS. When did you first reach that conclusion?
A. Actually, it was a conclusion based on animal studies that I did with Dr. Witschi and that were published under Dr. Rajini as the first author.
Q. In what year?
A. I don't recall, but probably 1994 to 1996, somewhere in that -- that period of time.
Q. And your opinion that ETS causes a decreased growth in the infant with maternal exposure to ETS is based primarily on studies that you've conducted?
A. It is based on that one animal study. I think it has really been confirmed, though, through epidemiological studies.

This was just one aspect of studies with environmental tobacco smoke that I personally had never really focused on, but because we were doing studies with environmental tobacco smoke and we could design a study using -- using rodents, I thought that it would be worthwhile to investigate, but I think the animal studies, although they may be helpful, I think the epidemiological literature is probably -- is stronger because there are just more studies, and they are bigger cohorts of -- you know, where they can look at differences in body weights of children to do these kind of comparisons.
Q. Now -- so for your opinion that ETS causes a decrease in... I guess we should say, "fetal growth"?
Would that be the right way to refer to the decrease in growth?
A. For our animal study, it was in fetal growth that we measured.
Q. So let me start that question again then.
For your opinion that ETS causes a decrease in fetal growth with maternal exposure to ETS, you're still relying primarily on epidemiology studies for that conclusion?
A. No, because we can't look at the fetus and do those kind of measurements in humans. It would have to be an animal study, and so that's only based on animal studies.
Q. You had talked about epidemiology you had

reviewed on that issue, though, and maybe I misunderstood you. What was the epidemiology discussing on the decreased growth?
A. The epidemiology has often looked at active smoking of the mother during pregnancy, and I think that's quite clear-cut that there is going to be a reduction in the birth weight of the child, but there have been other ongoing studies that have also tried to look at the effect of passive exposure during pregnancy to environmental tobacco smoke, and those studies have shown that there is a reduction in the birth weight of the child even with passive exposure to cigarette smoke.
Q. And you equate a reduction in birth weight with a reduction in fetal growth?
A. It would follow.
Q. Now, if -- okay. That answers that.
Let's go to decrements in pulmonary function

11:39:53 19 in children. When did you first reach the conclusion
11:39:56 20 that ETS caused decrements in pulmonary function in
11:40:05 21 children?
11:40:06 22 A. Again, this was during the literature review
11:40:08 23 of the epidemiology, so this would have been from that
11:40:14 24 period of 1991 through 1994.
11:40:22 25 Q. And that would be a literature review of the
11:40:27 26 epidemiologic literature?
11:40:29 27 A. Yes.
11:40:29 28 Q. So your opinion that ETS exposure causes

11:40:32 1 a -- causes decrements in pulmonary function in
11:40:36 2 children is based primarily on epidemiologic
11:40:39 3 literature?
11:40:40 4 A. Yes.
11:40:40 5 Q. And it's based on literature that you
11:40:42 6 reviewed as a result of funding you received from CIAR?
11:40:46 7 A. That's correct, but also by 1994, we were
11:40:50 8 receiving additional funding from another source.
11:40:54 9 Q. Okay. And again -- I'll do a lot of this
11:40:56 10 today. I should have asked this question earlier.
11:40:59 11 When we talked about the 1994 Rajini study
11:41:03 12 that you rely on for your opinion regarding decreased
11:41:06 13 fetal growth, first of all, that's the only animal
11:41:10 14 study you rely on for that opinion, correct?
11:41:15 15 A. That is correct.
11:41:15 16 Q. And that animal study you're relying on for
11:41:17 17 that opinion was a study you conducted under funding
11:41:20 18 from the CIAR, correct?
11:41:26 19 A. For part of that study, I was receiving
11:41:33 20 funding from the -- from CIAR. I do not recall if
11:41:37 21 Dr. Witschi was receiving funding for that particular
11:41:41 22 study in which he was the principal investigator for
11:41:45 23 that study.
11:41:47 24 Q. At a minimum, that study was in part funded
11:41:50 25 by CIAR. Would that be a fair way to say it?
11:41:54 26 A. Yes.
11:41:57 27 MR. KODSI: Okay. The videographer has told
11:42:03 28 me that we have probably now only about two minutes

11:42:03 1 left on the video record. So this is probably a good
11:42:06 2 time to take a break.
3 THE VIDEOGRAPHER: Thank you.
4 This marks the end of Tape Number 1 in the
11:42:08 5 deposition of Kent Pinkerton. Going off the record,
11:42:09 6 the time is 11:42.
7 (Recess taken)
12:03:01 8 (Mr. Cafferty is not present.)
12:03:44 9 THE VIDEOGRAPHER: Back on the record.
12:03:45 10 Here marks the beginning of Tape Number 2 in
12:03:48 11 the deposition of Kent Pinkerton. The time is 12:03.
12:03:53 12 MR. KODSI: Q. Dr. Pinkerton, one of the
12:03:57 13 diseases or health effects we were talking about
12:03:59 14 earlier was respiratory infection, and could you define
12:04:04 15 what you mean by "respiratory infection"?
12:04:12 16 A. I think this has to do with any kind of
12:04:14 17 bacterial or viral type of pathogen that leads to a
12:04:23 18 compromise in breathing, and it doesn't necessarily
12:04:30 19 just include only pneumonia but, I think, other
12:04:36 20 symptoms like flu like symptoms and things like that.
12:04:42 21 Q. Do you view ETS as a bacterial or a viral

12:04:46 22 pathogen?
12:04:48 23 A. No.
12:04:48 24 Q. Okay. So are there things other than
12:04:49 25 bacterial or viral pathogens that could compromise
12:04:53 26 breathing through respiratory infection?
12:04:59 27 A. I'm not sure I understand your question.
12:05:01 28 Q. Okay. Let me back up.

12:05:02 1 We were talking about your opinion that ETS
12:05:05 2 increases respiratory infection in children.
12:05:10 3 A. The risk of respiratory infection.
12:05:16 4 Q. In what way does ETS increase the risk of an
12:05:19 5 infection through a bacterial or viral agent?
12:05:24 6 A. If I understand your question, the thought
12:05:28 7 is that exposure to environmental tobacco smoke in some
12:05:33 8 way alters the immune function of the lung or some sort
12:05:39 9 of structural or functional parameter that may make it
12:05:44 10 more difficult to clear from the lungs or to neutralize
12:05:50 11 a bacterial or viral pathogen.
12:05:54 12 Q. Now, you had indicated that your opinion
12:05:59 13 that ETS increases the risk of respiratory infection is
12:06:04 14 based primarily on epidemiology studies?
15 A. Yes.
12:06:08 16 Q. Are epidemiology studies capable of
12:06:11 17 demonstrating alterations in the immune function of the
12:06:14 18 lung?
12:06:15 19 MR. BROOKEY: Objection; lacks foundation,
12:06:18 20 calls for speculation, but he can answer.
12:06:21 21 THE WITNESS: I think there are certain
12:06:22 22 parameters that can be used to see if there's a
12:06:25 23 compromise in immune function.
12:06:29 24 For example, one could look at the levels of
12:06:33 25 circulating antibodies. One could also look at the
12:06:39 26 function of cells, immune cells, either from the
12:06:45 27 respiratory system or circulating in the blood that
12:06:49 28 might be able to be an indicator of -- indication of

12:06:53 1 whether your immune function is appropriate at adequate
12:07:02 2 levels of protection or not.
12:07:05 3 MR. KODSI: Q. Let me ask you:
12:07:05 4 How does respiratory infection relate to
12:07:08 5 lung growth or development?
12:07:13 6 A. Only in an indirect way.
12:07:15 7 If you have sustained -- if you have a
12:07:18 8 respiratory infection during the time that your lungs
12:07:23 9 are growing and developing, that is a time in which
12:07:29 10 that type of infection could exacerbate or could
12:07:33 11 actually impair or in some way alter lung development.
12 (Mr. Cafferty rejoins the proceedings.)
12:07:47 13 MR. KODSI: Q. Okay. Now, talking about
12:07:48 14 each of -- let me go back again through each of the
12:07:58 15 diseases, and we'll start with respiratory infection.
12:08:02 16 Is ETS a necessary cause for nonsmokers to
12:08:05 17 develop respiratory infection? Do you understand that
12:08:10 18 question? Maybe I should --
12:08:13 19 A. Would you repeat it?
12:08:14 20 Q. Let me rephrase it.
12:08:16 21 In other words, do nonsmokers develop
12:08:19 22 respiratory infection without having been exposed to
12:08:22 23 ETS?
12:08:25 24 A. Yes.

12:08:25 25 Q. Could you list for me what the other causes
12:08:27 26 are for respiratory infection, to your knowledge?
12:08:36 27 A. Well, exposure to any type of infectious
12:08:40 28 agent. Typically, this is through inhalation, exposure

12:08:46 1 to someone else who has a respiratory infection, and it
12:08:52 2 is at that point in the disease that they are
12:08:56 3 contagious and can transmit the infection.
12:09:06 4 Q. I assume exposures to certain viruses.
12:09:09 5 A. Yes.
12:09:10 6 Q. Are there any in particular that are more
12:09:14 7 frequently associated with respiratory infection?
12:09:19 8 A. Not that I'm aware of.
12:09:21 9 Q. And you mentioned exposures to certain
12:09:23 10 bacteria.
12:09:25 11 A. Uh-huh.
12:09:26 12 Q. And --
12:09:27 13 A. Yes.
12:09:28 14 Q. -- are there any bacterias that you're aware
12:09:31 15 of that are particularly associated with increased risk
12:09:34 16 of respiratory infection?
12:09:35 17 A. Well, I think that there are both bacterial
12:09:38 18 as well as viruses that may -- that are more likely to
12:09:43 19 create a respiratory infection than others.
12:09:47 20 Q. Are there any that come to mind?
12:09:50 21 A. Not really. Pneumococcal would be one
12:09:56 22 example. The influenza virus would be an example.
12:10:03 23 Q. Okay. Now let's talk about increased asthma
12:10:07 24 in children. Do nonsmokers develop an increased risk
12:10:14 25 of asthma without ETS exposure?
12:10:21 26 A. I'm not sure I understand that question.
12:10:24 27 Q. Are there things other than ETS that can
12:10:26 28 result in an increased risk of asthma in nonsmokers,

12:10:31 1 other exposures that can result in that?
12:10:34 2 A. Yes. There are other things that can result
12:10:42 3 in asthma, yes.
12:10:42 4 Q. Okay. And what would those be?
12:10:42 5 A. Aeroallergens/pollens would be an example,
12:10:51 6 cockroach residues, house dust mites. There is also
12:11:07 7 some evidence to suggest that genetically one may be
12:11:12 8 exposed. One may be more -- I'm sorry. One may be
12:11:16 9 more susceptible or more likely to develop asthma based
12:11:21 10 on --
12:11:22 11 Q. You're talking --
12:11:23 12 A. -- a genetic basis.
12:11:25 13 Q. -- about a genetic predisposition to the
12:11:29 14 development of asthma?
12:11:29 15 A. Yes.
12:11:30 16 Q. Okay. Anything else you can think of?
12:11:32 17 A. Not at the moment.
12:11:33 18 Q. Okay. What about increased severity of
12:11:37 19 asthma in children, are there other factors other than
12:11:41 20 ETS, to your knowledge, that cause increased severity
12:11:46 21 of asthma in children?
12:11:50 22 A. Well, in terms of increased severity of an
12:11:53 23 asthmatic attack?
12:11:56 24 Q. Uh-huh.
12:11:57 25 A. Well, I think that there are probably a
12:11:59 26 number of factors that may be involved.
12:12:05 27 Again, potentially, exposure to

12:12:09 28 aeroallergens. There may be a predisposition based on

12:12:16 1 a stressful type of situation, would be examples that I
12:12:23 2 could think of.
12:12:24 3 Q. Would pollens also increase severity?
12:12:28 4 A. That would be included in aeroallergens.
12:12:31 5 Q. Yeah. All of the points that you just gave
12:12:32 6 me that might result in an increase in asthma, would
12:12:37 7 they also all result in an increase of the severity of
12:12:41 8 asthma?
12:12:42 9 A. There is the possibility that they could.
12:12:50 10 Q. Now, decreased fetal growth with maternal
12:12:54 11 exposure. Are there other things that a mother can be
12:12:58 12 exposed to that might result in decreased fetal growth?
12:13:03 13 A. Yes.
12:13:03 14 Q. And what might those be?
12:13:05 15 A. A lack of nutrition during pregnancy. There
12:13:14 16 is an undocumented -- so I don't know if it would
12:13:17 17 really count, but there is a report suggesting that
12:13:22 18 exposure to airborne particles may also lead to
12:13:28 19 decrements in fetal growth --
12:13:32 20 Q. Okay.
12:13:33 21 A. -- but I don't really know. That's really
12:13:35 22 in the peer-reviewed literature.
12:13:39 23 Q. And I think the last one is decrements in
12:13:42 24 pulmonary function. Are there other exposures that
12:13:44 25 might result in decrements in pulmonary function?
12:13:48 26 A. For children?
12:13:49 27 Q. For children.
12:13:54 28 A. Yes.

12:13:56 1 Q. What might those be?
12:13:58 2 A. Environmental air pollutants that include
12:14:08 3 ozone, nitrogen oxides, aerosols that may include
12:14:15 4 acidic aerosols.
12:14:19 5 Q. Anything else you can think of?
12:14:21 6 A. Not that I can think of.
12:14:30 7 Q. Okay. Now, with respect to your opinions
12:14:32 8 that ETS causes each of these health effects, do you
12:14:39 9 have in mind a particular concentration of ETS that can
12:14:43 10 cause these health effects? and we can walk through
12:14:46 11 each of them one at a time.
12:14:49 12 Let's start with respiratory infection.
12:14:51 13 Do you have in mind a particular level,
12:14:52 14 concentration, exposure, however you want to define it,
12:14:55 15 to ETS that would be responsible for increased risk of
12:14:58 16 respiratory infection?
12:15:00 17 A. No. I don't have any -- any level that I
12:15:06 18 would say would -- would be a threshold, if that is
12:15:11 19 what you're asking.
12:15:12 20 Q. Yes. Do you believe that there might be a
12:15:14 21 threshold?
12:15:17 22 A. From a scientific, well, basis, I don't know
12:15:24 23 of there being evidence for documentation of a
12:15:28 24 threshold for it.
12:15:30 25 Q. Do you have that belief for all health
12:15:31 26 effects or are there health effects where you believe
12:15:34 27 there might be a threshold?
12:15:36 28 A. Oh, I think there are certain things where

12:15:39 1 there may be a threshold.
12:15:41 2 Q. Are any of those health effects that we've
12:15:43 3 talked about today?
12:15:45 4 A. For respiratory infection? For
12:15:46 5 decrements --
12:15:48 6 Q. Yes.
12:15:48 7 A. -- in pulmonary function? I would think so,
12:15:56 8 that there may exist thresholds.
12:15:58 9 Q. Okay. So it's possible that there may exist
12:16:00 10 a threshold to ETS exposure below which there would not
12:16:06 11 be an increased risk of respiratory infection?
12:16:09 12 A. There is a possibility.
12:16:10 13 Q. That's not something you've studied?
12:16:11 14 A. No.
12:16:12 15 Q. And, in your opinion, that's not something
12:16:13 16 that the scientific community has reached consensus on?
12:16:18 17 A. Not that I'm aware of.
12:16:20 18 Q. Okay. And it's possible that there may be a
12:16:22 19 threshold below which ETS exposure does not cause an
12:16:25 20 increased risk in asthma?
12:16:30 21 A. There is a possibility.
12:16:30 22 Q. And that's not something that you have
12:16:32 23 studied?
12:16:35 24 A. Not to the degree that would lead me to an
12:16:38 25 answer.
12:16:39 26 Q. And, to your knowledge, that's not something
12:16:41 27 upon which the scientific community has reached
12:16:44 28 consensus on?

12:16:48 1 A. I'm not sure they've actually studied that.
12:16:52 2 Q. And the same question with respect to
12:16:54 3 increased severity of asthma. It's possible that there
12:16:58 4 might be a threshold below which exposure to ETS does
12:17:02 5 not result in an increased severity of asthma?
12:17:05 6 A. That is a possibility.
12:17:06 7 Q. And, again, it's not -- that's not something
12:17:08 8 that the scientific community has reached consensus on?
12:17:12 9 A. No.
12:17:13 10 Q. And it's also possible that there is a
12:17:16 11 threshold below which ETS exposure would not result in
12:17:23 12 decreased fetal growth?
12:17:23 13 A. That is true.
12:17:23 14 Q. And that's also not something upon which the
12:17:25 15 scientific community has reached consensus on?
12:17:29 16 A. I think there are a number of studies that
12:17:32 17 seem to suggest that there is a correlation between ETS
12:17:38 18 exposures and reduction in weight.
12:17:43 19 Q. Are there studies that address that from a
12:17:45 20 threshold perspective?
12:17:47 21 A. No.
12:17:48 22 Q. Do you think that the scientific community
12:17:51 23 has reached a consensus regarding whether there is a
12:17:54 24 threshold below which ETS exposure would not result in
12:17:57 25 decreased fetal growth?
12:18:00 26 A. I don't think there is.
12:18:02 27 Q. And then the last one would be decrements in
12:18:04 28 pulmonary function. Do you believe it's possible that

12:18:07 1 there is a threshold below which ETS exposure would not
12:18:10 2 result in decrements in pulmonary function?
12:18:13 3 A. Yes.

12:18:14 4 Q. And, again, that's not an issue upon which
12:18:16 5 the scientific community has reached consensus?
12:18:19 6 A. Again, I don't think they have.
12:18:41 7 MR. KODSI: Okay. Okay. Let's change focus
12:18:42 8 a little bit, and I wanted to walk through your
12:18:45 9 Curriculum Vitae with you briefly again, if you all
12:18:51 10 have copies of it. I've got one for -- one to mark.
12:18:55 11 Brian, do you want a copy?
12:18:57 12 MR. BROOKEY: Yes, please.
12:19:40 13 MR. KODSI: Can we go off the record for a
12:19:41 14 second?
12:19:42 15 THE VIDEOGRAPHER: Going off the record, the
12:19:44 16 time is 12:19.
17 (Discussion held off the record)
18 (Whereupon, Defendants' Exhibit 529 was
19 marked for identification.)
12:21:52 20 THE VIDEOGRAPHER: Back on the record, the
12:21:53 21 time is 12:21.
12:21:55 22 MR. KODSI: Q. Okay. Dr. Pinkerton, you've
12:21:57 23 been handed what has been marked as Exhibit 529.
12:22:00 24 That is your Curriculum Vitae, correct?
25 A. Yes.
12:22:02 26 Q. Is that the most current version --
12:22:04 27 A. Yes.
12:22:04 28 Q. -- of your Curriculum Vitae?

12:22:06 1 Is there anything that you need to add to it
12:22:08 2 to bring it up to date? and feel free to take whatever
12:22:13 3 time you need to...
12:23:06 4 A. This CV is complete with the exception of
12:23:10 5 abstracts published in the Year 2000.
12:23:15 6 Q. Okay. And you're looking at -- could you
12:23:17 7 read into the record the page you're looking at? I
12:23:19 8 think there's a number on the bottom.
12:23:22 9 A. The number is marked as PX-KEP-001011.
12:23:29 10 Q. And if you were to bring your CV up to date,
12:23:33 11 you would add a few items to that page?
12:23:36 12 A. Yes.
12:23:36 13 Q. Do you know, off the top of your head, what
12:23:38 14 those would be?
12:23:40 15 A. There are approximately seven abstracts that
12:23:46 16 were published for the American Thoracic Society
12:23:55 17 meeting that was held in May, in Toronto, Canada, and
12:23:59 18 these were published in the abstract booklets for
12:24:05 19 American Review of Critical Care Medicine, I think it's
12:24:15 20 called.
12:24:16 21 Q. Were any of those abstracts related issues
12:24:24 22 relevant to your testimony in this case?
12:24:26 23 A. No.
12:24:28 24 Q. Did any of them address tobacco smoke at
12:24:30 25 all?
12:24:31 26 A. Yes.
12:24:32 27 Q. Okay. Which ones addressed tobacco smoke?
12:24:34 28 A. The Effects of Environmental Tobacco Smoke

12:24:37 1 on Lung Development in Nonhuman Primates.
12:24:43 2 Q. But you don't intend on relying on that?
12:24:45 3 A. No.
12:24:46 4 Q. What did that show?
12:24:48 5 A. That there are significant effects of
12:24:52 6 perinatal exposure to environmental tobacco smoke in

12:24:58 7 Rhesus monkeys.
12:25:05 8 Q. I may be remembering incorrectly, but is
12:25:08 9 that part of the work that you discussed doing with
12:25:10 10 Dr. Slotkin?
12:25:14 11 A. No.
12:25:15 12 Q. Okay. I did remember incorrectly.
12:25:16 13 Were any of these seven abstracts work that
12:25:19 14 you are doing with Dr. Slotkin?
12:25:21 15 A. No.
12:25:23 16 Q. Okay. So as I'm looking through here, you
12:25:26 17 get to spend May in Toronto, Canada while Dr. Witschi
12:25:29 18 gets to spend June in Toledo, Ohio at the mouth/lung
19 symposium.
20 A. Yes.
12:25:34 21 Q. You think you got the better end of that
12:25:36 22 deal, right?
12:25:38 23 A. I think so.
12:25:39 24 Q. Were there any other papers related to
12:25:42 25 tobacco smoke other than the Rhesus monkey paper?
12:25:45 26 A. No. Correction.
12:25:48 27 Q. Oh.
12:25:50 28 A. A graduate student of mine presented the

12:25:52 1 effects of environmental tobacco smoke in mouse and
12:25:59 2 how it -- how it affects the response to subsequent
12:26:03 3 exposure to ozone.
12:26:07 4 Q. And, as you've already said, I assume that's
12:26:09 5 not a paper you're relying on for any opinions you
12:26:12 6 intend to offer in this case?
12:26:13 7 A. No.
12:26:13 8 Q. What did that paper show?
12:26:15 9 A. It showed that mice that are preexposed for
12:26:20 10 three days to environmental tobacco smoke develop an
12:26:24 11 acute sensitivity to the effects of ozone exposure, and
12:26:36 12 I should say, "acute." By "acute," I mean a
12:26:40 13 heighthened sensitivity to their effects to subsequent
12:26:44 14 exposure to ozone.
12:26:45 15 Q. Who is funding that ozone research?
12:26:48 16 A. That's funded by the Tobacco-related Disease
12:26:53 17 Research Program from the State of California.
12:26:57 18 Q. And the primate study that you mentioned,
12:27:00 19 who's funding that?
12:27:01 20 A. The California Tobacco-related Disease
12:27:05 21 Research Program.
12:27:05 22 Q. Are either of those studies receiving
12:27:07 23 funding from any private entity?
12:27:09 24 A. No.
12:27:15 25 Q. Okay. Other than the seven abstracts that
12:27:17 26 we've just -- oh, I'm sorry. Were those the only of
12:27:20 27 the seven related to tobacco smoke?
12:27:22 28 A. Yes.

12:27:23 1 Q. Well, briefly, why don't you just, if you
12:27:25 2 can, describe what the other five are, so...
12:27:31 3 A. Correction. There was one other abstract
12:27:32 4 from a collaborator, Dr. Laura VanWinkle, in which she
12:27:39 5 was examining the effects of environmental tobacco
12:27:44 6 smoke on injury repair in neonates of mice that had
12:27:51 7 been injured by -- through exposure to naphthalene.
12:28:06 8 Q. Okay. And who's funding that?
12:28:10 9 A. The California Tobacco-related Disease

Research Program.
Q. Any others?
A. I don't think so.
Q. Okay. Why don't we briefly, maybe, walk through what the other four are.
A. One abstract is dealing with a model in nonhuman primates of asthma in which the sensitivity is induced by a house dust mite allergen, and our studies were to look at changes in airway tone based on a special approach that we've developed in my lab of precision-cut lung slices in which we can actually measure changes in airway luminal size and actually correlate that by introducing increasing doses of methacholine, which is a -- causes bronchial constriction, and it's a classical pharmacological agent used to test the sensitivity of individuals to asthmatic light conditions --
Q. Okay.
A. -- and we correlated changes in airway

function to structural changes in the lungs of these -- in the airways of these lung slices that we prepared.
Q. And what about the others to the extent you can remember?
A. An abstract was presented on the effects of single versus mixed air pollutants on lung injury and pulmonary fibrosis. This was a study to examine the effects of ozone and nitrogen dioxide alone or in combination in the rat lung. These were studies that were done in adult animals and demonstrated that -- the role in which a growth factor, transforming growth factor beta, influences the degree of lung injury, how it's expressed over a period of time in which the injury and the pulmonary fibrosis, the results from exposure, is progressing.
Q. Now, is this a study looking for synergistic effects?
A. That -- interactive effects or synergistic effects.
Q. That's a good point.
Do you differentiate between "interactive effects" and "synergistic effects"?
A. We try to in all that we -- in all that we do.
Q. And what did you find in this particular study?
A. That there was actually a synergism between the two pollutants.

Q. And that was ozone and what else?
A. Nitrogen dioxide.
Q. Do you have those abstracts with you today?
A. No.
Q. Okay. Do you have them where you might be able to bring them tomorrow?
A. Yes.
MR. KODSI: If you're not going to object, I'm going to ask the doctor if he'd bring them with him tomorrow.
MR. BROOKEY: I'll take it under advisement.
MR. KODSI: Yeah. I just -- I know Brian

12:32:00 13 will know enough to anticipate that was coming, so...
12:32:03 14 Q. But, yeah, if you're comfortable with that,
12:32:04 15 I'd like to -- if you could bring those tomorrow...
12:32:07 16 A. Okay.
12:32:09 17 Q. Okay. Now we've talked about our abstracts.
12:32:12 18 Are there -- is there anything else that you
12:32:14 19 would add to your CV to bring it up to date?
12:32:17 20 A. No.
12:32:33 21 Q. Okay. Did I not get all seven? I'm being
12:32:36 22 told that I may not have asked you about all seven
12:32:39 23 abstracts. I've got one, two, three, four. Oh, I've
12:32:41 24 only got five. There are two others that we didn't
12:32:45 25 cover. Do you remember what those were?
12:32:47 26 We've got the three that address tobacco
12:32:50 27 smoke, one on asthma in nonhuman primates, and one on
12:32:54 28 the ozone and nitrogen dioxide.

12:32:58 1 A. Uh-huh. I don't remember at the moment. It
12:33:08 2 seems as though I had seven places to be, and so that's
12:33:11 3 why I...
12:33:14 4 Q. Seven different poster presentations to
12:33:17 5 make? Well, if you can bring those tomorrow, we can
12:33:22 6 see what the other two were, and if there's anything
12:33:25 7 worth talking about, we will.
12:33:27 8 A. Okay.
12:33:28 9 Q. Let me ask you this: Your CV then contains
12:33:31 10 all of your abstracts and publications.
12:33:34 11 A. With the exception of these that we've been
12:33:36 12 discussing.
12:33:37 13 Q. Sorry. Right, with the exception of those
12:33:39 14 seven. Do you have any publications where you have
12:33:42 15 reviewed epidemiologic literature? and let me make that
12:33:50 16 clear because I think that wasn't very clear.
12:33:53 17 Where the sole purpose of the publication
12:33:56 18 was to review epidemiologic literature.
12:34:00 19 A. That I wrote?
12:34:01 20 Q. Yes.
12:34:01 21 A. No.
12:34:02 22 Q. Have you ever authored an epidemiologic
12:34:05 23 study?
12:34:06 24 A. No.
12:34:07 25 Q. Have you ever been a principal investigator
12:34:09 26 on an epidemiologic study?
12:34:11 27 A. No.
12:34:11 28 Q. Or any type of investigator on an

12:34:13 1 epidemiologic study?
12:34:17 2 A. Yes.
12:34:18 3 Q. What would that be?
12:34:22 4 A. Now, it's unpublished, so does that mean no
12:34:26 5 in terms of...
12:34:26 6 Q. Well, let's talk about it. If you've been
12:34:29 7 an investigator on an epidemiologic study that's
12:34:32 8 unpublished, why don't we talk about that.
12:34:33 9 What would that study be?
12:34:34 10 A. It was a study in looking at young Hispanic
12:34:38 11 males from the Fresno County area and looking at the
12:34:43 12 potential for health effects associated with
12:34:46 13 occupational exposures to farming practices versus
12:34:51 14 other types of occupational exposures.
12:34:55 15 My role in this study was to serve as the

12:35:00 16 pathologist to look at autopsy cases from the medical
12:35:12 17 coroner's office, to examine for the presence of
12:35:15 18 mineral dusts or carbonaceous particulate matter and
12:35:21 19 for pathological changes that may be present within the
12:35:25 20 lungs.
12:35:27 21 Q. Would you view that? That sounds like
12:35:30 22 approaching a molecular epidemiology study.
12:35:34 23 Would you agree with that?
12:35:35 24 A. Yes.
12:35:36 25 Q. Most epidemiology studies don't actually
12:35:39 26 look at -- go as far as to look at the tissues of the
12:35:42 27 study.
12:35:43 28 A. That's correct.

12:35:43 1 Q. Now, you mentioned it was unpublished.
12:35:45 2 Is that research ongoing?
12:35:47 3 A. Yes, and it's also a submitted manuscript.
12:35:54 4 Q. Who was the lead or the principal
12:35:56 5 investigator on that study?
12:35:57 6 A. The principal investigator is Marc Schenker.
12:36:06 7 Q. And you said that has been submitted to a
12:36:09 8 journal?
12:36:09 9 A. Yes.
12:36:10 10 Q. Which journal?
12:36:11 11 A. {Environmental Health Perspectives.}
12:36:18 12 Q. And that's currently undergoing the peer
12:36:20 13 review process now?
12:36:22 14 A. Yes.
12:36:23 15 Q. And I assume that's not a study that you
12:36:24 16 intend on relying --
12:36:27 17 A. No.
12:36:27 18 Q. -- on for any opinions in this case?
12:36:29 19 Now, your role in that study, you indicated,
12:36:31 20 was as a pathologist. You didn't have any role as a
12:36:35 21 biostatistician --
12:36:37 22 A. No.
12:36:38 23 Q. -- in that study or you didn't do any
12:36:41 24 statistical analysis for that study?
12:36:43 25 A. I was involved with the statistical
12:36:44 26 analysis.
12:36:45 27 Q. What role did you have in the statistical
12:36:47 28 analysis?

12:36:48 1 A. In collecting the data, in organizing it in
12:36:52 2 a way in which it could be analyzed statistically, and
12:37:01 3 in providing that material to the biostatistician who
12:37:05 4 we consulted with, and we discussed the study design
12:37:11 5 and the parameters that would be reasonable for
12:37:16 6 comparison, that would be appropriate for comparison.
12:37:22 7 Q. Did you conduct any of the statistical
12:37:24 8 analysis for the study?
12:37:27 9 A. The actual running of the program?
12:37:29 10 Q. Yes.
12:37:30 11 A. No.
12:37:30 12 Q. You helped to prepare the data for someone
12:37:32 13 else to analyze?
12:37:34 14 A. That's correct.
12:37:34 15 Q. And you made recommendations as to how they
12:37:36 16 might look at the data?
12:37:38 17 A. That's correct.
12:37:38 18 Q. But there was a biostatistician that

12:37:40 19 actually went in and conducted the statistical
12:37:43 20 analysis?
12:37:43 21 A. That's correct. Uh-huh.
12:37:47 22 Q. Talk a little bit about what areas you
12:37:49 23 consider yourself to have expertise in. How would you
12:37:52 24 describe your area of expertise?
12:37:55 25 MR. BROOKEY: Objection; asked and answered.
12:37:58 26 He can answer it again. I'll also object to the extent
12:38:00 27 it's calling for a legal conclusion, but again, he can
12:38:04 28 describe his expertise.

12:38:05 1 THE WITNESS: Well, my expertise is in
12:38:10 2 environmental air pollutants and their effects on the
12:38:15 3 respiratory system. This includes being able to create
12:38:25 4 exposure conditions that would be well characterized
12:38:31 5 for selected environmental air pollutants that we can
12:38:34 6 generate in the laboratory. So this involves having
12:38:40 7 expertise in inhalation procedures and inhalation
12:38:45 8 toxicology. It also would involve looking at
12:38:52 9 parameters for understanding what's happening in the
12:38:59 10 respiratory system with growth and development as well
12:39:03 11 as with exposure to different types of environmental
12:39:06 12 factors that are primarily delivered to the respiratory
12:39:10 13 system by way of inhalation.
12:39:14 14 Although we are also -- I'm also involved in
12:39:16 15 studies that allow me to understand delivery of
12:39:22 16 different types of constituents that may be delivered
12:39:25 17 to the respiratory system, not by way of inhalation but
12:39:29 18 by way of the circulation.
12:39:33 19 My primary training is in doing pathological
12:39:37 20 assessment for lung injury and in -- extending that to
12:39:44 21 different disease processes that involve the
12:39:48 22 respiratory system and in doing quantitative analyses
12:39:54 23 rather than just simply subjective grading type of
12:39:58 24 criteria, using quantitative procedures that include
12:40:03 25 morphometry and stereology.
12:40:11 26 MR. KODSI: Q. How would you define
12:40:12 27 "morphometry"?
12:40:14 28 A. "Morphometry" is the -- basically, the

12:40:17 1 quantitative analysis of anatomical structures,
12:40:25 2 describing them in terms of cell numbers, volumes of
12:40:33 3 various cell compartments, surface areas that are --
12:40:39 4 and all in a quantifiable -- using numbers rather than
12:40:45 5 a grading scheme.
12:40:46 6 Q. And what was that, the stery...
12:40:48 7 A. Stereology.
12:40:49 8 Q. Stereology? What is "stereology"?
12:40:52 9 A. "Stereology" is also a quantitative approach
12:40:55 10 in which from two-dimensional profiles you derive
12:40:59 11 three-dimensional values. So this would be a form of
12:41:04 12 morphometry in which sections, tissue sections -- or
12:41:09 13 tissues are prepared and then can be sectioned and
12:41:13 14 viewed under a microscope, be it a dissecting
12:41:17 15 microscope, a light microscope or a transmission
12:41:21 16 electron microscope, and one can do analyses to use
12:41:25 17 those two-dimensional profiles to then derive
12:41:29 18 three-dimensional values for the whole organ system.
12:41:34 19 Q. Now, you're currently a professor at
12:41:38 20 California, Davis, correct?
12:41:41 21 A. Yes.

12:41:42 22 Q. What type of -- do you teach courses?
12:41:44 23 A. Yes.
12:41:44 24 Q. Could you describe for me the types of
12:41:45 25 courses you teach?
12:41:47 26 A. I teach courses in the School of Veterinary
12:41:53 27 Medicine. I teach cardiovascular anatomy to first-year
12:41:58 28 veterinary students that include the thoracic anatomy,

12:42:06 1 that involves the thoracic innervation, also the
12:42:12 2 circulation of the cardiovascular system as it is
12:42:18 3 important to the thoracic cavity.
12:42:22 4 I'm also involved in teaching respiratory
12:42:30 5 anatomy to first-year veterinary students, and this
12:42:30 6 involves both gross and microscopic training and
12:42:34 7 understanding of the respiratory system.
12:42:37 8 I'm also involved in teaching the
12:42:39 9 respiratory system anatomy to first-year medical
12:42:43 10 students at the University.
12:42:46 11 My responsibilities at the graduate level
12:42:50 12 involve teaching courses in the pharmacology and
12:42:54 13 toxicology graduate group, and my emphasis of teaching
12:42:58 14 there is in the area of inhalation toxicology and
12:43:03 15 understanding principles of particle deposition,
12:43:09 16 clearance, translocation, retention.
12:43:14 17 I'm also involved in the training of -- or
12:43:18 18 working with graduate students to prepare them for
12:43:21 19 taking their qualifying exams in preparation for their
12:43:27 20 doctoral degrees, which they must pass in order to
12:43:33 21 continue on in the graduate program.
12:43:37 22 I'm involved in actually being the major
12:43:42 23 professor, or mentor, for graduate students in a
12:43:46 24 variety of both masters and Ph.D. programs. My primary
12:43:51 25 focus has been graduate students who are getting their
12:43:55 26 degree in Pharmacology and Toxicology, but I also have
12:43:58 27 graduate students who will be getting their doctoral
12:44:03 28 degrees in Comparative Pathology as well as in

12:44:06 1 Immunology.
12:44:08 2 Q. Do you teach any graduate students who are
12:44:10 3 trying to obtain Ph.Ds in Epidemiology?
12:44:16 4 A. No, but I serve on a committee for
12:44:20 5 epidemiology graduate students.
12:44:23 6 Q. What type of committee is that?
12:44:24 7 A. It's for their qualifying exam. I have
12:44:28 8 served on one committee for a student who is getting
12:44:35 9 his Ph.D. in Epidemiology.
12:44:38 10 Q. Maybe I don't understand. When you say, "a
12:44:40 11 committee" --
12:44:42 12 A. Basic -- the committee, it's not a course,
12:44:48 13 but as graduate students complete their course work and
12:44:50 14 complete their preliminary studies for their Ph.D.,
12:44:56 15 then they must appear before a committee for their
12:44:59 16 qualifying exam.
12:45:00 17 Q. Is this to present their dissertation?
12:45:02 18 A. This is to present their proposal as well as
12:45:05 19 to defend in certain areas that include epidemiology
12:45:10 20 but also may include other areas that are relevant to
12:45:18 21 pursuing their research in epidemiology.
12:45:22 22 Q. But, just to make sure I understand, you
12:45:24 23 don't teach any courses in epidemiology?
12:45:26 24 A. That's correct.

12:45:26 25 Q. And you don't teach any courses in
12:45:28 26 biostatistics?
12:45:30 27 A. That's true.
12:45:30 28 Q. And do you teach any courses in statistics?

12:45:34 1 A. No, no formal courses.
12:45:43 2 Q. How are you chosen to be on a particular
12:45:46 3 committee with respect to a Ph.D. student?
12:45:50 4 A. For a qualifying exam?
12:45:52 5 Q. Yes, for a qualifying exam.
12:45:54 6 A. Actually, it's usually by assignment from
12:46:02 7 the Continuing Education Committee for any graduate
12:46:06 8 group, and so this is -- that's the typical way of
12:46:12 9 doing it. For the Epidemiology Group, because I'm not
12:46:16 10 a member of a graduate group, this was by special
12:46:20 11 request because of my expertise in lung pathology and
12:46:26 12 dealing with the farm worker issue that we're currently
12:46:30 13 studying.
12:46:31 14 Q. Was this particular epidemiology Ph.D.
12:46:33 15 candidate studying farm worker issues?
12:46:36 16 A. Yes.
12:46:36 17 Q. And that's how you were chosen on the
12:46:38 18 committee for him -- or her? I'm sorry.
12:46:42 19 A. For him.
12:46:43 20 Q. Oh, I got lucky.
12:46:46 21 A. And that would be my assumption.
12:46:52 22 Q. We were talking about the courses that you
12:46:53 23 teach. Do you teach any courses where you discuss
12:46:58 24 environmental tobacco smoke with your students?
12:47:03 25 A. There is a graduate course which is
12:47:07 26 specifically on respiratory toxicology in which I have
12:47:10 27 been asked to lecture on environmental tobacco smoke
12:47:13 28 in -- and its effects.

12:47:18 1 Q. Are there any textbooks that you are using
12:47:20 2 in that course on that issue?
12:47:22 3 A. No.
12:47:22 4 Q. What about -- well, why don't I be broader.
12:47:27 5 Are there any materials, written materials,
12:47:29 6 that you're going to prepare for that issue?
12:47:34 7 A. Typically, on an issue like that, we will
12:47:36 8 present them, perhaps, with a paper, a review paper
12:47:40 9 that they can -- can use for that particular lecture.
12:47:47 10 Q. Do you have a particular review paper in
12:47:49 11 mind that you intend to use?
12:47:51 12 A. I have used in the past the paper that
12:47:53 13 Dr. Witschi and Dr. Joad and I wrote, which was our
12:47:58 14 review of the health effects of environmental tobacco
12:48:02 15 smoke.
12:48:02 16 Q. Is that the 1997 paper titled "The
17 Toxicology of Environmental Tobacco Smoke"?
12:48:06 18 A. Yes.
12:48:07 19 Q. And that's the paper you would give your
12:48:09 20 students in that course?
12:48:10 21 A. For that lecture, yes.
12:48:12 22 Q. And that lecture is actually taking place in
12:48:14 23 the future, right? It hasn't taken place yet?
12:48:17 24 A. No. We've already -- the course is offered
12:48:20 25 every other year, and so I have taught that course
12:48:23 26 twice now.
12:48:25 27 Q. Okay. And when is the next time you

12:48:26 28 intend -- or that you know you're going to teach that

12:48:28 1 course?
12:48:30 2 A. This will probably be in 2002.
12:48:38 3 Q. So it will be way too ahead for me to ask
12:48:42 4 you if you intend to use that article in 2002, so why
12:48:45 5 don't I backtrack and ask you when the most recent time
12:48:48 6 you taught that course was.
12:48:50 7 A. It was this year.
12:48:55 8 Q. 2000 or 1999?
12:48:57 9 A. I think it was 1999.
12:48:59 10 Q. And when you taught that course -- and let
12:49:03 11 me backtrack. When you taught that course on
12:49:05 12 respiratory pathology and you addressed ETS issues in
12:49:15 13 1999 --
12:49:15 14 A. Uh-huh.
12:49:15 15 Q. -- the paper you chose to provide your
12:49:15 16 students with was the 1997 review conducted by
12:49:15 17 yourself, Dr. Witschi and Dr. Pinkerton -- I mean
12:49:19 18 Dr. Joad.
12:49:20 19 A. Uh-huh. Yes.
12:49:22 20 Q. Okay. And that paper is titled "The
12:49:23 21 Toxicology of Environmental Tobacco Smoke"?
12:49:27 22 A. Yes.
12:49:28 23 MR. KODSI: Do you have that? Okay. Good.
12:49:30 24 I'm just going to go ahead and find it real
12:49:32 25 quick to mark it so that we know we're talking about
12:49:34 26 the same paper.
27 (Whereupon, Defendants' Exhibit 530 was
28 marked for identification.)

12:49:58 1 MR. KODSI: Q. I've just handed you
12:49:59 2 Exhibit 530, Doctor. Is that the paper that we were
12:50:04 3 just discussing that you gave your students in 1999
12:50:07 4 when you were lecturing to them about ETS issues?
12:50:13 5 A. Yes.
12:50:16 6 Q. Are there any other papers that you gave
12:50:18 7 them to review?
12:50:19 8 A. Not that I recall.
12:50:37 9 Q. Okay. Are there any other courses in which
12:50:38 10 you've addressed environmental tobacco smoke with your
12:50:42 11 students?
12:50:49 12 A. I teach an undergraduate course in air
12:50:58 13 pollution. I'm just one of many faculty members who
12:51:01 14 teach in that course, and my primary responsibility
12:51:05 15 there is to talk about the anatomy of the respiratory
12:51:11 16 system and potential environmental impacts on the
12:51:14 17 respiratory system, and there is the possibility that I
12:51:18 18 have discussed with undergraduate students the effects
12:51:22 19 of environmental tobacco smoke on the respiratory
12:51:24 20 system, but that was not the major focus of that
12:51:28 21 lecture.
12:51:29 22 Q. Were there any materials you provided to
12:51:30 23 your students --
12:51:33 24 A. Specifically on --
12:51:33 25 Q. -- specifically for that issue, on the ETS
12:51:35 26 issue in that class?
12:51:36 27 A. No.
12:51:38 28 Q. Okay. Now, we talked early on today about

12:51:42 1 that you feel you have some expertise in epidemiology
12:51:45 2 as it pertains to your animal studies, but I just want
12:51:48 3 to clear up a few things.
12:51:50 4 Do you consider yourself to be an
12:51:51 5 epidemiologist?
12:51:54 6 A. No.
12:51:58 7 Q. Do you recognize there are Ph.D. degrees in
12:52:00 8 Epidemiology?
12:52:02 9 A. Yes.
12:52:02 10 Q. And there are Ph.D. degrees in
12:52:04 11 Biostatistics?
12:52:05 12 A. Yes.
12:52:05 13 Q. And you don't have either of those degrees,
12:52:08 14 correct?
12:52:08 15 A. No.
12:52:12 16 Q. Okay. Have you ever served as a -- well,
12:52:14 17 let me back -- start that one over. Do you serve as a
12:52:18 18 peer reviewer for any scientific journals?
12:52:21 19 A. Yes.
12:52:21 20 Q. Which ones would those be?
12:52:25 21 A. {American Journal of Pathology}, {American
12:52:41 22 Journal of Physiology - Lung Cellular and Molecular
12:52:48 23 Physiology}, {Journal of Applied Physiology}, {American
12:53:00 24 Journal of Respiratory Cell and Molecular Biology},
12:53:14 25 {Experimental Lung Research - Gerontology.}
12:53:37 26 Those are the main ones I can think of.
12:53:38 27 Q. Are you on the editorial boards for any of
12:53:41 28 those journals?

12:53:41 1 A. I was just released from the editorial board
12:53:44 2 from {American Journal of Physiology - ... Cellular and
12:53:49 3 Molecular Physiology}.
12:53:51 4 Q. Are there any others?
12:53:52 5 A. No.
12:53:52 6 Q. And are there any that you're on the
12:53:55 7 editorial boards for that we didn't just talk about?
12:53:57 8 A. No. Did I say, {Environmental Health
12:54:00 9 Perspectives}?
12:54:01 10 Q. I don't believe you did. Are you on the
12:54:02 11 editorial board for {Environmental Health
12 Perspectives}?
12:54:04 13 A. Oh, no, just a reviewer.
12:54:07 14 Q. So you've never served as a peer reviewer
12:54:09 15 for an epidemiologic journal, correct?
12:54:14 16 A. That's correct.
12:54:16 17 Q. And you've never served as a peer reviewer
12:54:19 18 on an epidemiology study?
12:54:23 19 A. That's correct.
12:54:40 20 MR. KODSI: I think I'm about to change
12:54:41 21 topics, and it's almost 1:00 o'clock. This might be a
12:54:44 22 good time rather than go into something new.
12:54:47 23 THE VIDEOGRAPHER: Going off the record, the
12:54:48 24 time is 12:54.
12:54:52 25 (Afternoon recess taken 12:54 to 1:59)
26 (Mr. Cafferty and Ms. Moore are not
27 present.)
28

1 AFTERNOON SESSION - 1:59 PM
13:59:31 2 THE VIDEOGRAPHER: Back on the record, the
13:59:32 3 time is 1:59.

CONTINUED EXAMINATION BY MR. KODSI

MR. KODSI: Q. We were -- I think where we left off, Dr. Pinkerton, we were going through your Curriculum Vitae, and I just had a few more follow-ups on that, and not that you would have this in your CV, but have you given any lectures or presentations on ETS-related issues other than the ones we've talked about at the University?

A. I've been asked to speak at national meetings on environmental tobacco smoke.

Q. Could you give me examples of some of those?

A. The first time was at an annual meeting for the Society of Toxicology, in which the topic was "Societal and Environmental Issues of Environmental Tobacco Smoke," and actually, that was published as a paper.

Q. And that presentation resulted in a paper in which you, Dr. Witschi, Gio Gori, Chris Coggins, and others -- I can't remember. You may be able to remember who the others are -- had a peer review paper --

A. Yes.

Q. -- on the results of that symposium?

A. Yes.

Q. And that was sponsored by the Society for

Toxicology?

A. Yes.

Q. And that was 1994?

A. I don't recall. It could have been.

Q. Within a few years of that?

A. Uh-huh. Right. 1994 or 1995.

(Ms. Moore rejoins the proceedings.)

MR. KODSI: Q. Any other presentations on ETS?

A. I presented this year at the American Thoracic Society meeting a talk on lung growth, lung development. It was titled -- the symposium was titled Nature versus Nurture, and I gave the talk on the impact of perinatal exposure to environmental pollutants on lung development, and that talk included, in large measure, work that involves environmental tobacco smoke, and that was presented in May of this year in Toronto.

Q. And that was part of the meeting we talked about earlier that you attended in Toronto, right?

A. Of the various abstracts.

Q. Right. The American Thoracic Society?

A. Yes. Uh-huh.

(Mr. Cafferty rejoins the proceedings.)

MR. KODSI: And let me mark that.

(Whereupon, Defendants' Exhibit 531 was marked for identification.)

MR. KODSI: Q. Dr. Pinkerton, you've been

handed -- I'll give you time to put your glasses on.

You've been handed what has been marked as Exhibit 531. Is that the Symposium Overview that we were just discussing that you coauthored with Drs. Witschi, Coggins, Gio Gori, and Arthur Penn, which is the name I couldn't remember?

14:03:26 7 A. Yes.
14:03:27 8 Q. Could you explain for me how -- was this
14:03:32 9 a -- was this paper drafted as a joint effort by those
14:03:36 10 authors or could you describe for me how this paper was
14:03:41 11 drafted?
14:03:42 12 A. It was spearheaded by Dr. Witschi, who was
14:03:46 13 the chair of the symposium, and he requested of each of
14:03:52 14 the speakers that they would provide a summary of what
14:03:56 15 they felt was the most important issues relative to
14:04:01 16 their topic of discussion.
14:04:04 17 Q. How was the abstract prepared, if you know?
14:04:09 18 A. The abstract was prepared by Dr. Witschi.
14:04:15 19 Q. And, in preparing this publication, did the
14:04:17 20 authors review each other's summary or was it an
14:04:21 21 independent effort on behalf of each of the authors?
14:04:29 22 A. I don't recall. I know that we were -- we
14:04:34 23 did certainly review our portions of the contribution
14:04:38 24 as well as the abstract, the introduction and overview
14:04:42 25 for the paper, but I don't recall whether I was asked
14:04:48 26 to have any input in the other coauthors'
14:04:55 27 contributions.
14:04:55 28 Q. Do you recall asking any of the other

14:04:57 1 authors to have any input into your contribution?
14:05:04 2 A. Only Dr. Witschi --
14:05:07 3 Q. And --
14:05:07 4 A. -- that I recall.
14:05:08 5 Q. Did this paper undergo any form of peer
14:05:10 6 review other than review by the named authors?
14:05:17 7 A. My understanding is that there was some peer
14:05:20 8 review process since this was in {Fundamental and
14:05:26 9 Applied Toxicology}, but I may be wrong on that.
14:05:29 10 Q. Your understanding is based on the fact that
14:05:31 11 {Fundamental and Applied Toxicology} is a peer review
14:05:35 12 journal?
14:05:35 13 A. That's correct.
14:05:39 14 Q. Now, we were talking about lectures and
14:05:41 15 presentations you've given on environmental tobacco
14:05:43 16 smoke, and we've talked about the Society for
14:05:46 17 Toxicology presentation and the one at the Thoracic
14:05:50 18 Society. Are there any others that you can think of?
14:06:00 19 A. Not that I can think of off the top of my
14:06:02 20 head. I know that we've presented at a national
14:06:06 21 meeting posters and that -- so -- of dealing with
14:06:12 22 environmental tobacco smoke and that we have presented
14:06:18 23 the work on how preexposure to short-term duration of
14:06:25 24 environmental tobacco smoke sensitizes the lungs to the
14:06:30 25 effects of ozone was presented as a platform
14:06:37 26 presentation at last year's meeting for the American
14:06:43 27 Thoracic Society in San Diego.
14:06:46 28 Q. Now, you had mentioned a national meeting

14:06:50 1 that you had just done a presentation of.
14:06:54 2 Where was that national meeting?
14:06:55 3 A. This was for the American Thoracic Society.
14:06:57 4 Actually, it was -- it was actually an international
14:07:00 5 meeting.
14:07:01 6 Q. Is this still the one in Toronto --
14:07:02 7 A. Yes.
14:07:03 8 Q. -- that we talked about earlier?
14:07:06 9 A. (Nods head.)

14:07:08 10 Q. Oh, okay. Have you been invited -- or have
14:07:12 11 you given any presentations to any governmental bodies
14:07:16 12 on environmental tobacco smoke?
14:07:22 13 A. The workshop that was organized last fall by
14:07:24 14 the US Environmental Protection Agency was a
14:07:29 15 presentation to invited scientists, which had in the
14:07:35 16 audience some members of the Environmental Protection
14:07:40 17 Agency, but it was not a formal presentation to that
14:07:44 18 agency but rather as part of the workshop sponsored by
14:07:49 19 the US EPA.
14:07:55 20 Q. Now, what was the purpose of that workshop?
14:07:57 21 A. To examine critical windows of exposure for
14:08:01 22 children's health that involved the major organ
14:08:06 23 systems, including the respiratory system, immune
14:08:10 24 system and others.
14:08:11 25 Q. So that workshop wasn't an ETS workshop.
14:08:13 26 It's just that the issues you discussed were in some
14:08:16 27 way related to ETS?
14:08:19 28 A. That's correct.

14:08:21 1 Q. Have you ever testified before a government
14:08:24 2 body on topics related to ETS?
14:08:28 3 A. No.
14:08:29 4 Q. You've never testified before the Federal
14:08:34 5 Occupational Safety and Health Administration?
14:08:34 6 A. No.
14:08:35 7 Q. What about the California Environmental
14:08:37 8 Protection Association --
14:08:38 9 A. No.
14:08:38 10 Q. -- Agency?
14:08:39 11 A. No.
14:08:40 12 Q. No? Okay.
14:08:42 13 Have you participated in any legislative
14:08:45 14 activity related to environmental tobacco smoke or
14:08:49 15 smoking restrictions?
14:08:51 16 A. No.
14:08:51 17 Q. How about any regulatory activity?
14:08:56 18 A. No.
14:08:57 19 Q. Are you affiliated with any public interest
14:08:59 20 groups that address tobacco smoke issues?
14:09:03 21 A. No.
14:09:11 22 Q. Okay. Let's talk a little bit about the
14:09:13 23 Center for Indoor Air Research, which we've agreed to
14:09:17 24 refer to as "CIAR." You're familiar with CIAR?
14:09:20 25 A. Yes.
14:09:21 26 Q. Could you describe generally what you know
14:09:23 27 about the makeup of CIAR?
14:09:27 28 A. Well, it was an agency that was established

14:09:34 1 to fund independent research in issues having to do
14:09:41 2 with indoor air pollution, based in Maryland. They
14:09:48 3 followed a protocol in establishing their center that
14:09:57 4 was similar to the Health Effects Institute, which was
14:10:01 5 established for studies on environmental air pollution
14:10:07 6 and automobile emissions, things like that, as an
14:10:12 7 independent agency.
14:10:14 8 So that was my impression is that CIAR was
14:10:18 9 following that as their template for trying to
14:10:22 10 establish a similar agency for studying indoor air
14:10:27 11 pollution.
14:10:27 12 Q. And the Health Effects Institute -- why

14:10:32 13 don't you describe for me what the Health Effects
14:10:34 14 Institute is.
14:10:35 15 A. It is an independent agency that is funded
14:10:40 16 half by the US Environmental Protection Agency and half
14:10:44 17 by sponsors in the -- from the automotive industry.
14:10:51 18 Q. And, in fact, some of the research that
14:10:54 19 you've done on ozone has been funded by the Health
14:10:57 20 Effects Institute, correct?
14:10:58 21 A. That's correct.
14:10:59 22 Q. Is your current work on ozone that you
14:11:02 23 discussed among the seven abstracts for Toronto
14:11:05 24 receiving funding by the Health Effects Institute?
14:11:08 25 A. No.
14:11:15 26 Q. Now, you said it is an organization set up
14:11:18 27 for independent research. What did you mean by
14:11:22 28 "independent research"?

14:11:25 1 A. Basically research that would not be done
14:11:31 2 within the -- that agency but would be funded by having
14:11:39 3 a request for proposals submitted and then go through a
14:11:43 4 peer review process and selected for funding. So I
14:11:49 5 would assume that most of this research would be done
14:11:52 6 based on ideas that originated with the investigators
14:12:00 7 that -- where CIAR might have had certain special
14:12:03 8 topics of interest that they would like to see
14:12:06 9 proposals submitted for, but it would be completely
14:12:11 10 independent that the investigators that formulated
14:12:14 11 those proposals and submitted them that they would be
14:12:17 12 their own ideas, what they thought would be relevant
14:12:20 13 for study in those particular topics that were
14:12:25 14 suggested by CIAR.
14:12:28 15 Q. Now, you said, "I ... assume," and I just
14:12:31 16 want to make sure I understand your answer.
14:12:33 17 Based upon your experiences with the CIAR,
14:12:36 18 do you have any reason to believe that there were
14:12:38 19 studies done by investigators that were not the
14:12:41 20 original idea of the investigator?
14:12:44 21 A. No.
14:12:45 22 Q. All of the studies that you did that were
14:12:47 23 funded by CIAR were your original ideas?
14:12:51 24 A. That's correct.
14:12:51 25 Q. And you submitted proposals to CIAR based
14:12:53 26 upon those ideas?
14:12:54 27 A. Yes.
14:12:57 28 Q. What is your understanding of what the

14:12:59 1 "member organizations" of the CIAR were?
14:13:06 2 A. After receiving funding and getting more and
14:13:09 3 more information about the Center for Indoor Air
14:13:13 4 Research, I found that about 80 percent of the
14:13:16 5 sponsorship was from the tobacco industry, from tobacco
14:13:24 6 companies sponsoring funds for research.
14:13:31 7 Q. And did you find that out after the first
14:13:32 8 time you had received funding?
14:13:37 9 A. As I recollect, I think as I submitted the
14:13:41 10 proposal that the sponsors -- well, actually, after I
14:13:47 11 got the funding, I became aware as I got more
14:13:51 12 information where the funding was -- sources were
14:13:54 13 coming from.
14:13:57 14 Q. And you continued to conduct studies under
14:13:59 15 CIAR funding even after you had knowledge that 80

14:14:01 16 percent of the money was coming from the tobacco
14:14:07 17 companies; is that correct?
14:14:08 18 A. Yes.
14:14:09 19 Q. In your opinion, what percentage of your
14:14:10 20 work related to environmental tobacco smoke was funded
14:14:13 21 by CIAR?
14:14:15 22 A. Up to date?
14:14:17 23 Q. Up to date.
14:14:23 24 A. Approximately 25 percent.
14:14:41 25 Q. And I don't know how long this will take, so
14:14:45 26 I'll ask you, if you think it's going to take a while,
14:14:48 27 maybe we can do it during a break, but could you go
14:14:55 28 through your Curriculum Vitae, which I think is

14:14:55 1 Exhibit -- you have it in front of you --
14:14:56 2 A. Uh-huh.
14:14:56 3 Q. -- 529 and identify for the record which
14:15:05 4 papers in there were done with CIAR funding?
14:15:11 5 A. Okay. By number?
14:15:14 6 Q. Probably the easiest way to do it would be
14:15:16 7 to refer to -- since you've got different sections, if
14:15:20 8 you're going through the Abstract Section, let us know
14:15:22 9 it's abstract and then give us the number, and then
14:15:24 10 when you go through the -- however you've got it
14:15:26 11 divided up.
14:15:27 12 A. Okay.
14:15:28 13 Q. You're now in the Publications section,
14:15:29 14 which is on Page PX-KEP-0000983. So why don't you
14:15:38 15 identify by number the ones on those section.
14:15:42 16 A. Okay. Publication 23, Publication 25,
14:16:16 17 Publication 27, Publication 32, but this was probably
14:16:36 18 under Witschi's proposal. I don't know if he would
14:16:41 19 have acknowledged my proposal. Publication 33,
14:16:56 20 Publication 37, which is Exhibit Number 531.
14:17:01 21 Dr. Witschi did not wish to acknowledge support from
14:17:05 22 any sources of funding on this particular paper.
14:17:10 23 Q. Let me just make sure I understand.
14:17:11 24 Was that at the request of the CIAR or was
14:17:15 25 that Dr. Witschi's own decision?
14:17:17 26 A. That was Dr. Witschi's own decision.
14:17:19 27 Q. Were there funding sources other than CIAR
14:17:22 28 related to Exhibit 531?

14:17:25 1 A. In 1994, we were beginning to receive
14:17:29 2 funding from the California Tobacco-related Disease
14:17:34 3 Research Program, so this is where there might be some
14:17:37 4 overlap.
14:17:38 5 Q. And is your funding from that California
14:17:42 6 program indicated anywhere in Exhibit 531?
14:17:49 7 A. No.
14:17:49 8 Q. So there are no funding sources identified
14:17:51 9 in that exhibit?
14:17:52 10 A. That's correct.
14:17:54 11 Q. You had left off on Publication 37.
14:18:06 12 A. From this point on, the publication
14:18:10 13 acknowledgments for sources of funding will be both
14:18:13 14 CIAR as well as the California Tobacco-related Disease
14:18:20 15 Research Program.
14:18:21 16 Publication 38. Again, I'll just say
14:18:35 17 Publication 39, which is a Witschi publication. I
14:18:40 18 don't know for sure if he acknowledged CIA support or

14:18:46 19 not -- CIAR support. Publication 40, 41, 42, 43.
14:19:08 20 Publication 47 again is a Witschi, and I don't know if
14:19:13 21 he would have -- although I am the first author, he was
14:19:17 22 the corresponding author. Publication 48, 49, 51, 53.
14:19:50 23 Publication 54 I don't think it acknowledged, but it
14:19:55 24 would have uncovered --
14:19:57 25 Q. There are a couple of times we've talked
14:19:59 26 about publications where you're not sure they would
14:20:01 27 have acknowledged CIAR funding, but you think they
14:20:03 28 received some. What would be the basis for not

14:20:05 1 acknowledging CIAR funding?
14:20:07 2 MR. BROOKEY: Objection; calls for
14:20:09 3 speculation. He can answer.
14:20:18 4 THE WITNESS: My thought might be -- my
14:20:21 5 thought is that it is a part of a review, and
14:20:26 6 therefore, some of the things in the review have
14:20:30 7 already been acknowledged, and therefore, Dr. Witschi
14:20:36 8 did not wish to acknowledge it again.
14:20:39 9 MR. KODSI: Q. Was there any time during
14:20:41 10 the course that we talk about papers that might not
14:20:44 11 acknowledge CIAR funding that that decision was based
14:20:49 12 on a request from CIAR?
14:20:51 13 A. No.
14:20:52 14 Q. So every time that we've talked about a
14:20:54 15 paper that might not acknowledge CIAR funding, that was
14:20:58 16 solely the decision of the author?
14:21:00 17 A. That's correct.
14:21:01 18 Q. Okay. I just wanted to make sure that we
14:21:03 19 understood that.
14:21:07 20 A. Uh-huh.
14:21:08 21 Q. We stopped off at 54.
14:21:14 22 A. At this point, we're in 1997, and the
14:21:18 23 funding from CIAR had been completed.
14:21:49 24 Publication 70. I would have to look at
14:21:52 25 that to see if that has CIAR acknowledgment or not, but
14:21:58 26 it would certainly have tobacco -- the Tobacco-related
14:22:04 27 Disease Research Program support acknowledged.
14:22:09 28 Publication 73, Publication 76.

14:22:47 1 Q. And I think you had mentioned
14:22:49 2 Publications 70, 73 and 76 were completed after the
14:22:53 3 CIAR funding was finished?
14:23:00 4 A. Yes.
14:23:00 5 Q. But were those publications still based on
14:23:04 6 data that was generated under CIAR funding?
14:23:07 7 A. Yes.
14:23:19 8 Q. Okay.
14:23:19 9 A. And I think that is it. There is a
14:23:21 10 possibility that Publication 82, which is the most
14:23:26 11 recent publication, might have CIAR acknowledgment, but
14:23:34 12 it's -- the bulk of the funding came from the
14:23:37 13 California Tobacco-related Disease Research Program.
14:23:56 14 Now, abstracts.
15 Q. Yes.
14:23:57 16 A. Do you want to go through those?
14:23:59 17 Q. Let's, for the record, identify the page.
14:24:01 18 The abstract begins on page...
14:24:04 19 A. PX-KEP-000995.
14:24:10 20 Q. Okay. Walk through which of those numbers
14:24:12 21 have CIAR funding.

14:24:14 22 A. Okay. Abstract 31, 33, 39, 43, 44, 48, 54,
14:25:11 23 55, 61, 63, 64, 65, 66, 68, 79, 80, 89, 90, 93, 95, 97,
14:27:01 24 99, 112, 115. We're now into that period where there's
14:27:47 25 no longer CIAR funding, but 117 may have had some
14:27:51 26 support, 122, 124, 125, and I think at that point I'd
14:28:21 27 say there might be some additional ones --
14:28:24 28 Q. Okay.

14:28:25 1 A. -- but...
14:28:35 2 Q. And that's it for the CV -- right? --
14:28:38 3 A. Right.
14:28:41 4 Q. -- with the publications and abstracts?
14:28:43 5 A. Right.
14:28:43 6 Q. Now, earlier when you talked about 25
14:28:45 7 percent of your work being funded by CIAR, is that 25
14:28:50 8 percent -- I'm sorry. Let's back up because that's a
14:28:53 9 misleading question.
14:28:54 10 25 percent of your work related to ETS has
14:28:58 11 been funded by CIAR. Are you talking about as compared
14:29:02 12 with all the other funding sources or were you saying
14:29:05 13 that 25 percent of your papers have received CIAR
14:29:08 14 funding?
14:29:11 15 A. That is based on sources only for
14:29:16 16 environmental tobacco smoke studies.
14:29:21 17 Q. We've listed -- and my math may be off a
14:29:24 18 little bit if I've missed a number, but approximately
14:29:26 19 22 publications and 28 abstracts that were at least in
14:29:31 20 part based on CIAR funding, does that constitute only
14:29:36 21 25 percent of your publications on ETS or would the
14:29:39 22 number actually be higher?
14:29:42 23 A. No. Actually, that constitutes more than 25
14:29:46 24 percent of my publications on ETS.
14:29:49 25 Q. About what percentage would you say it
14:29:50 26 encompasses?
14:29:52 27 A. On ETS, at this point in time, that is
14:29:57 28 probably 75 percent.

14:30:01 1 Q. So whereas maybe 25 percent of your
14:30:08 2 ETS-related funding has come from CIAR, 75 percent of
14:30:14 3 the papers you have published have some sort of CIAR
14:30:16 4 funding. Is that the right way to say it?
14:30:19 5 A. Actually, as I think about this, because of
14:30:21 6 the last two years of funding from the California
14:30:28 7 Tobacco-related Disease Research Program in which we've
14:30:29 8 had probably close to 15 abstracts published -- and
14:30:40 9 that just continues to keep growing, that perhaps the
14:30:45 10 number of publications with CIAR funding would -- 75
14:30:50 11 percent may be an overestimate --
14:30:52 12 Q. Okay.
14:30:52 13 A. -- of that, so...
14:30:54 14 Q. It would be somewhere between 50 and
14:30:56 15 75 percent?
14:30:57 16 A. That would be correct.
14:31:04 17 Q. Now, although CIAR funded those
14:31:08 18 publications, you did all of the research, correct?
14:31:13 19 A. That's correct.
14:31:14 20 Q. And the work was your own, and you were free
14:31:16 21 to publish whatever results you obtained?
14:31:19 22 A. Yes.
14:31:20 23 Q. In fact, CIAR encouraged you to publish?
14:31:24 24 A. Yes.

14:31:27 25 Q. Are there any unpublished papers or studies
14:31:30 26 that you have that have been funded by CIAR?
14:31:34 27 A. Yes.
14:31:34 28 Q. What would those be?

14:31:37 1 A. These are a number of studies in which we
14:31:40 2 looked at lower concentrations of environmental tobacco
14:31:45 3 smoke than we've been publishing at. We also -- so
14:31:52 4 that was a very extensive study, a dose response study
14:31:56 5 that was funded by CIAR. We've also done a number of
14:32:03 6 studies to look more specifically about the epithelial
14:32:09 7 maturation, differentiation between fetal lung
14:32:13 8 development with maternal exposure to ETS with regard
14:32:18 9 to neuroendocrine cells. That was supported by CIAR.
14:32:28 10 Q. Anything else?
14:32:32 11 A. Those are the major things that we have not
14:32:36 12 published.
14:32:37 13 Q. Now let's talk first about the lower
14:32:39 14 concentration studies. Why are those not published?
14:32:46 15 A. Their manuscripts are still in preparation.
14:32:52 16 Q. Are you going to be relying on that
14:32:53 17 unpublished data for any opinions you intend to offer
14:32:56 18 in this case?
14:32:58 19 A. No, not unless it's asked if we've measured
14:33:03 20 effects at lower concentrations than in our published
14:33:07 21 results.
14:33:19 22 Q. And the epithelial maturation and lung
14:33:22 23 development research, why has that not been published?
14:33:27 24 A. Again, it's a paper in preparation or
14:33:29 25 actually two papers in preparation.
14:33:35 26 Q. And with respect to those papers that are in
14:33:38 27 preparation for both the lower concentrations and the
14:33:40 28 epithelial maturation, CIAR has encouraged you to

14:33:44 1 publish those results as well?
14:33:46 2 A. Since they no longer exist, they are not
14:33:49 3 encouraging or discouraging.
14:33:51 4 Q. That's fair. At the time that you had done
14:33:53 5 these studies originally, CIAR was still in existence?
6 A. Yes.
14:33:57 7 Q. And at the time that they were still in
14:33:58 8 existence, they were at least encouraging you to
14:34:01 9 publish those results?
14:34:02 10 A. Uh-huh. Yes.
14:34:04 11 Q. Have there ever been any results you've
14:34:06 12 obtained that CIAR has tried to discourage you from
14:34:10 13 publishing?
14:34:11 14 A. No.
14:34:20 15 Q. Now, the research -- I'm assuming then that
14:34:22 16 the research for these two unpublished manuscripts was
14:34:26 17 conducted pre 1997?
14:34:29 18 A. Yes, and part of those studies have
14:34:36 19 continued on since 1997.
14:34:46 20 Q. Who did you correspond with primarily at
14:34:50 21 CIAR?
14:34:52 22 A. Lynn Channing.
14:34:57 23 Q. Did you ever work with any -- who was Lynn
14:35:01 24 Channing?
14:35:01 25 A. She was our project manager for our contract
14:35:04 26 with CIAR.
14:35:07 27 Q. She's not a scientist?

14:35:09 28 A. She has a Ph.D.

14:35:11 1 Q. Does she? She's just not a name I'm
14:35:15 2 familiar with.
14:35:20 3 A. I think she also goes by her maiden name,
14:35:28 4 Lynn Kosak-Channing.
14:35:28 5 Q. That last part sounded more familiar. Okay.
14:35:30 6 Did you work with any scientists that are
14:35:32 7 affiliated with CIAR?
14:35:38 8 A. In doing my research? In doing my
14:35:41 9 experiments?
14:35:41 10 Q. Yes.
14:35:42 11 A. No.
14:35:44 12 Q. Okay.
14:35:45 13 A. But they did sponsor a workshop to help us
14:35:49 14 get started on our research.
14:35:52 15 Q. Why don't you describe that workshop for me.
14:35:55 16 A. It was in the first year of our funding. It
14:35:58 17 was held in Maryland, and they -- people at CIAR
14:36:09 18 brought in scientists from around the country who had
14:36:14 19 either experience in studying environmental tobacco
14:36:18 20 smoke or measuring it or generating it under
14:36:22 21 experimental conditions. They also brought in
14:36:26 22 individuals who had experience in measuring biomarkers
14:36:30 23 for exposure to environmental tobacco smoke, and they
14:36:35 24 brought in a scientist who was very familiar with doing
14:36:41 25 small rodent studies, inhalation studies.
14:36:44 26 Q. Do you remember the names of any of the
14:36:46 27 scientists that were there?
14:36:47 28 A. A number of them. Not all of them, but...

14:36:50 1 Q. Why don't we go through some of the names
14:36:52 2 that you remember that attended that.
14:36:53 3 A. Okay.
14:36:54 4 Q. You referred to that as a workshop?
14:36:56 5 A. It was a workshop.
14:36:57 6 Q. Okay.
14:36:57 7 A. Uh-huh.
14:36:58 8 Q. And when was that?
14:36:59 9 A. That would have been, I believe, 1990 or
14:37:04 10 1991. Really, the purpose of it was to make sure
14:37:10 11 that -- I think there were two groups that were funded,
14:37:13 12 that I recall, and it was just to help us to make sure
14:37:17 13 that when we started doing these experiments -- since
14:37:21 14 for me I had never been involved with studies with
14:37:24 15 environmental tobacco smoke -- that we would do them in
14:37:28 16 a manner that would be reproducible and that would be
14:37:33 17 characterized.
14:37:35 18 Q. Right. And you found that workshop to be a
14:37:37 19 valuable experience for helping you to understand
14:37:39 20 environmental tobacco smoke?
14:37:41 21 A. That's right.
14:37:41 22 Q. And that workshop helped to develop the
14:37:45 23 general scientific understanding of environmental
14:37:49 24 tobacco smoke?
14:37:49 25 A. That's correct.
14:37:49 26 Q. And the workshop that was funded by CIAR
14:37:53 27 helped to further scientific understanding in the
14:37:57 28 community about environmental tobacco smoke by helping

14:38:01 1 to make you understand it better?
14:38:03 2 A. Certainly, for those who were in attendance
14:38:05 3 who were going to engage in scientific research, it was
14:38:09 4 very helpful.
14:38:11 5 Q. Why don't you start by describing the
14:38:14 6 scientists that attended.
14:38:16 7 A. Well, the scientist who helped us with small
14:38:20 8 animal studies and inhalation systems was Joe Mauderly
14:38:26 9 from the Lovelace Inhalation Tox -- Inhalation
14:38:31 10 Institute, Respiratory Institute.
14:38:34 11 Q. And that Lovelace is in New Mexico?
14:38:38 12 A. Yes. Also invited was Neal Benowitz, from
14:38:43 13 UC San Francisco, to talk to us about characterization
14:38:49 14 of exposures, biomarkers of exposure -- nicotine/
14:38:54 15 cotinine measurements.
14:38:59 16 I'm not going to remember her last name, but
14:39:01 17 it was Nancy from the American Health Foundation was
14:39:05 18 there who --
14:39:08 19 Q. Haley?
14:39:09 20 A. Nancy Haley, who had experience in working
14:39:13 21 with exposing volunteers to environmental tobacco smoke
14:39:21 22 and how you did analyses on that type of study if you
14:39:30 23 were going to be using human volunteers.
14:39:37 24 We had a person there who was very familiar
14:39:42 25 with field monitoring for environmental tobacco smoke
14:39:46 26 as well as generation of environmental tobacco smoke
14:39:48 27 under experimental conditions. His name is Roger
14:39:52 28 Jenkins.

14:39:54 1 Q. And he's with the Oak Ridge National
14:39:57 2 Laboratories?
14:39:57 3 A. Yes.
14:39:57 4 Q. And do you recognize Dr. Jenkins as an
14:39:59 5 expert on ETS exposures?
14:40:01 6 MR. BROOKEY: Objection to the extent it
14:40:02 7 calls for a legal conclusion, but he can answer.
14:40:06 8 THE WITNESS: Dr. Jenkins was very
14:40:06 9 instrumental in helping us to set up our inhalation
14:40:10 10 facility at UC Davis, and the type of direction that he
14:40:16 11 gave to us was extremely helpful in helping us to be
14:40:23 12 able to do sound and logical experiments with animals.
14:40:34 13 MR. KODSI: Q. Okay. Anyone else that you
14:40:36 14 remember?
14:40:44 15 A. Not at the moment. I'm sure there were
14:40:47 16 more, but...
14:40:48 17 Q. Right. Well, let me ask this:
14:40:50 18 Just from going through the list, it looks
14:40:52 19 like this -- let's see if you agree with me -- that
14:40:55 20 this CIAR workshop to discuss ETS, they certainly
14:41:00 21 didn't invite only tobacco industry employees to come
14:41:03 22 and make a presentation to you, correct?
14:41:06 23 A. That's correct.
14:41:06 24 Q. In fact, a lot of the people they invited to
14:41:09 25 talk about ETS are -- were affiliated with public
14:41:14 26 health organizations.
14:41:15 27 MR. BROOKEY: Objection. It's vague and
14:41:16 28 ambiguous, but he can answer.

14:41:20 1 THE WITNESS: That was my impression is that
14:41:21 2 they all had experience in public health or in academic
14:41:29 3 settings.

14:41:35 4 MR. KODSI: Q. And what was your
14:41:35 5 understanding of -- I think you've already answered
14:41:38 6 that, so let me withdraw that.
14:41:39 7 What was your overall impression of the
14:41:49 8 Center for Indoor Air Research as a funding
14:41:52 9 organization?
14:41:58 10 A. My impression is that they were interested
14:42:02 11 in helping us to get started in our research if we
14:42:08 12 were -- if we received funding and that they were
14:42:17 13 conscientious in helping us to go forward with that
14:42:22 14 research, but they were not intrusive in any way in the
14:42:28 15 type of research we were doing.
14:42:31 16 Q. When you say, "not intrusive," in other
14:42:34 17 words, you did your own research. No strings attached?
14:42:42 18 A. That's right.
14:42:42 19 Q. And you've received funding from
14:42:42 20 organizations other than the CIAR, correct?
14:42:43 21 A. That's correct.
14:42:44 22 Q. As we talked about earlier, you've
14:42:46 23 received -- your ozone studies had been funded by the
14:42:49 24 EPA and automobile industry?
14:42:54 25 A. (Nods head.) Well, the Health Effects
14:42:56 26 Institute, uh-huh.
14:42:57 27 Q. Right.
14:42:57 28 A. Uh-huh.

14:42:58 1 Q. And you've had studies on tobacco smoke
14:43:00 2 funded by organizations other than the CIAR?
14:43:03 3 A. That's correct.
14:43:03 4 Q. In fact, I think there was a period of time
14:43:07 5 when you indicated that some of your studies were being
14:43:09 6 funded by the CIAR and the California tobacco-related
14:43:14 7 disease program?
14:43:16 8 A. That's correct.
14:43:18 9 Q. During the time where you had studies funded
14:43:20 10 by both CIAR and the California tobacco-related disease
14:43:30 11 program, how would you compare the conduct of the CIAR
14:43:33 12 with the conduct of the California tobacco-related
14:43:37 13 disease program?
14:43:37 14 MR. BROOKEY: Objection; lack of foundation,
14:43:40 15 irrelevant, vague and ambiguous.
14:43:42 16 You can answer.
14:43:44 17 THE WITNESS: I thought that they both
14:43:48 18 provided us with directions. In some ways, I thought
14:43:53 19 that the Center for Indoor Air Research was not as
14:44:01 20 demanding in terms of deadlines for reports as it would
14:44:05 21 be through the state-funded program.
14:44:08 22 MR. KODSI: Q. And when you provided
14:44:10 23 reports to the state-funded program, were those reports
14:44:20 24 reviewed by the program and you got comments back?
14:44:24 25 A. No.
14:44:29 26 Q. Now, have you ever done any research for an
14:44:33 27 organization other than CIAR where you were asked not
14:44:36 28 to publish your results?

14:44:38 1 A. No.
14:44:42 2 Q. Have you ever done any research for an
14:44:43 3 organization other than CIAR where you were asked to
14:44:46 4 maybe change or alter any of your results?
14:44:49 5 A. No.
14:44:55 6 Q. And my understanding is, at least it's your

opinion, that the conduct of CIAR in no way deviated from the conduct you're used to having when you're dealing with funding organizations?

A. That's correct.

Q. Now, we mentioned, too, that CIAR no longer exists, but if CIAR did exist today, would you have any concerns about doing studies funded by CIAR?

MR. BROOKEY: Objection; lacks foundation, calls for speculation, but he can answer.

THE WITNESS: I would -- I guess I would have some reservations based on my experience of finding that there are certain scientific journals who refuse to publish if funding comes through sources that are thought to be supported by the tobacco industry.

MR. KODSI: Q. Let's talk about that a little bit. What experiences have you had?

A. With the American Thoracic Society, I've adamantly opposed their policy of -- basically their attitude of saying that if any type of scientific research has any kind of funding that can be directed back towards the tobacco industry that they will not review it and will not publish it, and I have found

that to be a very illogical policy that they've made.

Q. Why did you feel that that's illogical?

A. Because I think that science needs to stand up on its own without -- without being influenced as to the source of the funding. I think the science can be judged on its merits alone through a peer review process.

Q. And your experience was that the American Thoracic Society refused to publish one of your papers because it received tobacco funding?

A. That is correct. They refused to review it.

Q. They wouldn't even look at it?

A. (Nods head.)

Q. And the only reason they gave you for not wanting to look at it was because it had been funded by CIAR?

A. That's correct.

Q. Are there any other organizations or journals that you're aware of that have that policy?

A. I think I've heard rumors of that, but I'm not aware of any other journals like that.

Q. Do you know the names of the ones you think you've heard?

A. No.

Q. But you have heard that there are others?

A. Potentially, yeah.

MR. KODSI: Okay. Let me mark this one, and Brian, I just realized he -- let's go off the record

for just one second.

THE VIDEOGRAPHER: Going off the record, the time is 2:48.

(Discussion held off the record)

THE VIDEOGRAPHER: Back on the record, the time is 2:48.

(Whereupon, Defendants' Exhibit 532 was marked for identification.)

MR. KODSI: Q. I've handed you what has

been marked as Exhibit 532, and let me just tell you this is Exhibit B to your declaration which we marked as Exhibit 528, if I'm right.

Is your declaration Exhibit 528?

A. Yes.

Q. Okay. So Exhibit 532, actually, was part of Exhibit 528, correct?

A. I assume so. Uh-huh.

Q. Okay. I think the other part was your CV that we've also gone through, which is Exhibit 529.

I just wanted to ask you why you attached this document -- well, first of all, let me ask you what "Exhibit B" is.

A. "Exhibit B" is our executed copy of our contract from the Center for Indoor Air Research, funding our first contract through the Center for Indoor Air Research.

Q. And are you relying on Exhibit B for any opinions you intend to offer in this case?

A. No.

Q. Why was Exhibit B attached to your declaration?

A. Because this funding came from the Center for Indoor Air Research, and typically, the contracts that we have, they are to the University of California, but we are -- we have the stewardship over making sure that the research of that contract is done and just to make sure that there was no potential problems with my preparing a declaration based on the fact that I had -- a good portion of that research had been funded from the Center for Indoor Air Research. That was the reason for attaching it --

Q. Okay.

A. -- just so that everyone would know where the funding came from and if there was any legal stipulations about -- about how I could use that research, those research findings.

Q. Because the -- you said, I think, the majority of the research that you cite in your declaration was based on CIAR funding?

A. A good portion of it.

Q. And Exhibit B just provides the contract under which that funding took place?

A. That's correct.

Q. Okay.

A. Uh-huh.

MR. KODSI: Actually, although it's not

quite an hour, I think I might be able to cut out some stuff from my outline, so why don't we take a five-minute break, and maybe it will go a lot quicker.

THE VIDEOGRAPHER: This marks the end of Tape Number 2 in the deposition of Kent Pinkerton.

Going off the record, the time is 2:52.

(Recess taken)

(Messrs. Cafferty and Lendrum are not present.)

THE VIDEOGRAPHER: Back on the record.

Here marks the beginning of Tape Number 3 in the deposition of Kent Pinkerton. The time is 3:07.

15:07:56 13 MR. KODSI: Q. Okay. When we were talking
15:07:58 14 about CIAR, Dr. Pinkerton, you mentioned two
15:08:02 15 manuscripts that are in the works but haven't been
15:08:06 16 published yet that relate to CIAR funding, and I wanted
15:08:09 17 to probably probe those in a little more detail.
15:08:12 18 The first one you mentioned looked at lower
15:08:14 19 concentrations of ETS. Why don't you tell me what -- a
15:08:19 20 little bit about that study.
15:08:20 21 A. Okay. We have established that we were
15:08:23 22 measuring effects at the particulate level of
15:08:27 23 1 milligram per cubic meter, and so our interest was to
15:08:30 24 see if we would be able to detect any effects at lower
15:08:36 25 concentrations of the particulate matter or we just
15:08:41 26 referred to it as the Total Suspended Particulate, or
15:08:45 27 TSP.
15:08:46 28 So the design of the study was to do

15:08:48 1 simultaneous exposures at .1, .3, .5, and 1.0 milligram
15:08:58 2 per cubic meter of TSP in animals beginning at birth
15:09:03 3 through approximately 100 to 120 days of age and to
15:09:15 4 determine if we would see any effects on lung
15:09:21 5 development or cellular maturation or biochemical
15:09:27 6 metabolic expression in the respiratory system.
15:09:35 7 Q. Okay. And what did you find?
15:09:37 8 A. We found that there were measurable effects
15:09:41 9 based on enzyme activity measurements as low as
15:09:47 10 .3 milligrams per cubic meter of TSP and that there
15:09:54 11 were actually some alterations in cell expression at
15:10:03 12 0.1 milligram per cubic meter but that those were not
15:10:11 13 measured as something being statistically significant
15:10:17 14 that we could demonstrate that -- without question that
15:10:19 15 there was an effect at that level.
15:10:23 16 Q. What do you mean by "statistically
15:10:25 17 significant" there?
15:10:27 18 A. That the chances of that being just by
15:10:29 19 random occurrence were less than 1 in 20.
15:10:43 20 Q. So the 59 percent confidence level?
15:10:46 21 A. That's correct. Yeah. So we were using a
15:10:48 22 P value of less than or equal to .05.
15:10:55 23 Q. Now, you indicated that you had -- you
15:10:59 24 exposed -- and when we say, "animals," are we talking
15:11:02 25 about rats?
15:11:03 26 A. Yes.
15:11:03 27 Q. Which species?
15:11:05 28 A. These are Sprague-Dawley strain rats.

15:11:12 1 Q. So you had five exposure groups including a
15:11:17 2 control group?
15:11:18 3 A. That's correct.
15:11:19 4 Q. So you had one group that was exposed to
15:11:21 5 just fresh air, one group that was exposed to
15:11:26 6 100 micrograms per cubic meter total suspended
15:11:31 7 particles, one group exposed to 300 micrograms, one
15:11:35 8 group exposed to 500 micrograms, and one group exposed
15:11:38 9 to 1,000 micrograms or 1 milligram?
15:11:42 10 A. That's correct.
15:11:47 11 Q. And the lowest exposure level at which you
15:11:49 12 found a statistically significant effect was at the
15:11:54 13 group exposed to 300 micrograms?
15:11:58 14 A. That's correct.
15:11:58 15 Q. And in the group exposed to a concentration

15:12:00 16 of 100 micrograms per cubic meter TSP, you did not find
15:12:04 17 any statistically significant differences between that
15:12:08 18 group and the control group?
15:12:09 19 A. That's correct. We were able to make
15:12:20 20 observations that there was a difference between the
15:12:22 21 two, but based on animal to animal variability, we
15:12:28 22 would not venture to say that we were measuring an
15:12:31 23 effect.
24 (Mr. Cafferty rejoins the proceedings.)
15:12:34 25 MR. KODSI: Q. Now, what is the effect that
15:12:36 26 you saw at 300 micrograms per cubic meter?
15:12:40 27 A. This was a change in the expression for
15:12:43 28 metabolic function in the respiratory tract of these

15:12:50 1 animals for metabolizing constituents that could be
15:12:54 2 inhaled into the lungs. Specifically, we were
15:13:01 3 measuring the Cytochrome P450 mono-oxygenase system and
15:13:06 4 looking at the IA1 isozyme.
5 (Mr. Lendrum rejoins the proceedings.)
15:13:10 6 MR. KODSI: Q. What is the significance of
15:13:11 7 that effect with respect to health effects?
15:13:19 8 A. We don't have a direct answer for that.
15:13:25 9 All we know is that the perinatal exposure
15:13:29 10 to environmental tobacco smoke caused an alteration
15:13:34 11 from what we typically would see in an animal that
15:13:38 12 would just be exposed to air.
15:13:41 13 Q. Now you said, "perinatal." Was this study
15:13:44 14 strictly postnatal?
15:13:45 15 A. You're correct, and I should be corrected in
15:13:47 16 that. It was only a postnatal study for this
15:13:54 17 CIAR-funded program.
15:13:58 18 Q. Did you find -- now, the effects that you
15:14:00 19 found at 300 micrograms per cubic meter TSP, those were
15:14:06 20 statistically significant?
15:14:07 21 A. That's correct.
15:14:09 22 Q. Did you find effects at 500 micrograms per
15:14:13 23 cubic meter?
15:14:14 24 A. Yes.
15:14:15 25 Q. Were they any different from the effects
15:14:16 26 that you found at 300 micrograms per cubic meter?
15:14:20 27 A. They were higher. They were greater.
15:14:22 28 Q. More effects, but were there any different

15:14:25 1 types of effects that you found?
15:14:26 2 A. No. We were seeing the same effects
15:14:29 3 occurring in both the airways of the lungs as well as
15:14:33 4 in the gas exchange portions of the lungs.
15:14:36 5 Q. Were you strictly looking for changes in
15:14:41 6 expression of metabolic function in this study?
15:14:44 7 Was that the only end point you were looking
15:14:45 8 for?
15:14:46 9 A. We were also interested in looking at
15:14:48 10 potential structural changes in terms of development of
15:14:53 11 the lungs and -- but we only looked at the end point,
15:15:00 12 which was 100 and -- well, it basically ranged from 100
15:15:06 13 to 120 days, and we did not see any structural changes
15:15:11 14 due to exposure to environmental tobacco smoke at any
15:15:16 15 of those concentrations.
15:15:18 16 Q. So you looked for potential structural
15:15:19 17 changes in the lung, but for all the concentrations you
15:15:23 18 used in this study, you couldn't find any?

15:15:26 19 A. We could not find any structural
15:15:28 20 alterations.
15:15:29 21 Q. What else -- was there anything else you
15:15:31 22 looked for?
15:15:35 23 A. In these studies, we also would look at
15:15:40 24 cellular expression for certain proteins in addition to
15:15:47 25 the Cytochrome P450 isozymal system.
15:15:52 26 Q. And I know you're going to have to spell
15:15:54 27 that for the court reporter after the next break.
15:16:00 28 A. Okay.

15:16:00 1 Q. What other cellular expressions did you look
15:16:03 2 for?
15:16:04 3 A. Markers of maturation, such as expression of
15:16:10 4 proteins within airway epithelial cells.
15:16:20 5 Q. And what did you find when you looked for
15:16:21 6 those markers of maturation?
15:16:23 7 A. In animals that were 120 days of age, there
15:16:28 8 was no significant differences based on -- compared to
15:16:32 9 control animals.
15:16:33 10 Q. For any of the exposure groups?
15:16:34 11 A. For any of the exposure groups.
15:16:37 12 Q. And maybe this is another one I should have
15:16:39 13 asked earlier, but when we talk about animals that have
15:16:41 14 made it to 120 days, do you have an equivalent time
15:16:47 15 period in humans that that would represent?
15:16:49 16 In other words, if an animal makes it to 120
15:16:52 17 days, is there a particular human age that that would
15:16:55 18 be representative of?
15:16:56 19 A. That would probably be equivalent to
15:16:59 20 adolescence, to a teenager.
15:17:02 21 Q. To a teenager. So at least 13 years of
15:17:04 22 age --
15:17:05 23 A. Uh-huh.
15:17:05 24 Q. -- for a human child?
15:17:08 25 A. Uh-huh.
15:17:11 26 Q. Is there anything else you looked for in
15:17:13 27 that study?
15:17:13 28 A. No, not with CIAR funding, and actually, I

15:17:21 1 should say that, no, there were not any other things
15:17:23 2 that we did with that study.
15:17:27 3 Q. Okay. I guess I'm not sure I understand it.
15:17:30 4 You were inclined, at least initially,
15:17:32 5 maybe, to differentiate between the CIAR-funded portion
15:17:35 6 and some other portion of the study?
15:17:38 7 A. Right. Actually, what we have continued to
15:17:40 8 study is more of the physiological changes as well as
15:17:46 9 some of the cellular changes of the airways, but those
15:17:50 10 studies are based on -- because we can only afford to
15:17:54 11 do one -- one concentration for our studies, we've
15:17:58 12 opted to stay with the 1 milligram per cubic meter
15:18:03 13 concentration for those studies.
15:18:09 14 Q. Now, you indicated when we first talked
15:18:11 15 about this unpublished paper that you weren't relying
15:18:14 16 on it for any of the opinions you intend to offer at
15:18:17 17 trial. Why were you not going to rely on it for any
15:18:23 18 opinions?
15:18:23 19 A. Because it's not undergone peer review
15:18:25 20 evaluation.
15:18:34 21 Q. Is it undergoing peer review now?

15:18:35 22 A. No. It's still a paper in preparation.
15:18:39 23 Q. So when you said it was in manuscript form,
15:18:41 24 you haven't submitted it to a journal yet?
15:18:43 25 A. That's correct.
15:18:44 26 Q. Is it in a manuscript form that you'd be
15:18:46 27 willing to share with us?
15:18:52 28 A. Well, since it's not peer-reviewed, it's

15:18:55 1 still not at that point where it really should be
15:19:00 2 disseminated, but there are some indications of those
15:19:03 3 results found in the abstracts as well as in the review
15:19:08 4 paper that is marked Exhibit 530.
15:19:15 5 Q. Okay. Let's talk about the abstracts first.
15:19:19 6 Are there abstracts I could look at to have
15:19:21 7 a better understanding of that data? Would they be
15:19:24 8 cited in your CV?
15:19:26 9 A. Yes.
15:19:26 10 Q. Okay. If you could point out the particular
15:19:28 11 abstracts that would give us a better understanding of
15:19:32 12 that study...
15:19:45 13 A. The first author is Lee, L-e-e, so if you do
15:19:50 14 see it before me...
15:19:54 15 Q. Oh.
15:20:00 16 A. Probably Abstract Number 97. I think that
15:20:05 17 is it.
15:20:14 18 Q. Are there any others or would it just be
15:20:16 19 that one?
15:20:18 20 A. That's the primary one.
15:20:19 21 Q. Okay.
15:20:20 22 A. Uh-huh.
15:20:20 23 Q. And what about number -- no? Okay.
15:20:26 24 Now -- oh. If you're still looking at that,
15:20:29 25 I don't want to interrupt you. Do you think there
15:20:32 26 might be more?
15:20:32 27 A. No. No.
15:20:33 28 Q. Okay. You had mentioned in one of the

15:20:34 1 papers we've already marked, which is exhibit -- for
15:20:38 2 the record so we know which exhibit you're looking at.
15:20:40 3 A. Exhibit Number 530.
15:20:42 4 Q. Yes. If you could identify what pages in
15:20:44 5 Exhibit 530 discuss this data...
15:20:59 6 A. It is contained -- the exposure conditions
15:21:02 7 are contained in Table 1 of the paper, which is found
15:21:08 8 on Page 33 for -- and it just shows the exposure
15:21:18 9 conditions for particulate matter, for nicotine and the
15:21:22 10 carbon monoxide levels for that study, which is listed
15:21:27 11 as Reference 107 in Table 1, and then in Table 2, the
15:21:38 12 studies that are again listed as Reference 107 that
15:21:43 13 talk about exposure concentrations of .3 and
15:21:49 14 .5 milligrams TSP per meter cubed also refer to the --
15:21:57 15 those studies.
15:22:00 16 Q. Did you say just the .3 and .5?
15:22:02 17 A. That's correct.
15:22:03 18 Q. Where does the .1 come from in Table 2 on
15:22:07 19 Page 34?
15:22:08 20 A. I think that is a study that was by
15:22:12 21 Dr. Coggins. Yes. By Dr. Coggins, Aries, Mosberg, and
15:22:27 22 colleagues --
15:22:29 23 Q. Okay.
15:22:29 24 A. -- in 1993.

15:22:31 25 Q. And I noticed that the -- under References
15:22:34 26 for the .3 and .5 exposure group in Table 2, you
15:22:40 27 reference Number 107, which is the Lee abstract.
15:22:44 28 Is that the same Lee abstract that we just

15:22:46 1 found in your CV? Just to make sure there's not
15:22:48 2 another one out there, if you wouldn't mind
15:22:51 3 double-checking that for me. It's hard to tell because
15:23:15 4 the page numbers are a little different, but I think
15:23:19 5 it's --
15:23:19 6 A. Yeah, I think it is the same. I think that
15:23:21 7 the A14, 18 as listed in my CV, is the abstract number;
15:23:27 8 whereas, this is the --
15:23:28 9 Q. Page number.
15:23:29 10 A. -- page number. Uh-huh.
15:23:59 11 Q. Now, we've talked about one of the two
15:24:02 12 unpublished CIAR papers. The other one is on
15:24:06 13 epithelial maturation?
15:24:08 14 A. Right.
15:24:08 15 Q. What was the -- why don't you describe that
15:24:11 16 study for me.
15:24:13 17 A. One of our observations with the increased
15:24:16 18 airway hypersensitivity with exposure to environmental
15:24:23 19 tobacco smoke was a significant increase in the number
15:24:26 20 of neuroendocrine cells that were found in the airways
15:24:31 21 of these same animals that had been tested, and so our
15:24:35 22 question that we wished to address, because they were
15:24:38 23 elevated only to a statistically significant degree in
15:24:46 24 the lungs of rats that had been exposed in utero and
15:24:49 25 postnatally to environmental tobacco smoke -- we wished
15:24:52 26 to address were these neuroendocrine cells actually
15:24:56 27 changing in terms of their frequencies during fetal
15:25:00 28 development if the mother was being exposed to

15:25:02 1 environmental tobacco smoke. So that was the first
15:25:06 2 study. Those -- again, it's a paper in preparation.
15:25:12 3 The findings of those studies show that
15:25:15 4 there is highly significant increases in the numbers of
15:25:18 5 neuroendocrine cells found in both the future central
15:25:22 6 airways of these fetal lungs as well as the peripheral
15:25:26 7 airways.
15:25:27 8 And then we took that one step further
15:25:30 9 because neuroendocrine cells are such rare epithelial
15:25:34 10 types within the airways. They represent a very small
15:25:38 11 fraction of the total epithelial population that we
15:25:42 12 wanted to look three-dimensionally at the airway tree,
15:25:47 13 at least the main airway path in one lobe as a way of
15:25:51 14 trying to say, well, does exposure to environmental
15:25:55 15 tobacco smoke alter the numbers of these neuroendocrine
15:25:59 16 cells, and so that's a second paper that's in
15:26:01 17 preparation, but that was only looking for these
15:26:04 18 neuroendocrine cells in the postnatal period, at 7 days
15:26:10 19 of age and 21 days of postnatal age.
15:26:15 20 Q. And what dose groups did you use in that
15:26:17 21 study?
15:26:17 22 A. 1 milligram per cubic meter.
15:26:19 23 Q. And that's the only group?
15:26:21 24 A. That's correct.
15:26:40 25 Q. Okay. Let's switch topics a little bit, and
15:26:42 26 I just want to talk about just some -- what I'd call
15:26:44 27 fundamental principles of ETS exposure and toxicology,

just to make sure we're all on the same page.

First of all, let me ask you this: How do you define "environmental tobacco smoke"?

A. "Environmental tobacco smoke" is the combination of sidestream cigarette smoke coming off of the smoldering end of a cigarette as well as exhaled mainstream cigarette smoke from individuals who are actively smoking.

Q. Is environmental tobacco smoke more dilute than sidestream smoke?

A. For who? I mean...

Q. In real world environments where humans are exposed to environmental tobacco smoke.

A. Uh-huh. Well, basically if we're talking about exposures and -- an active smoker will be taking in far more particulate and vapor and gas phase constituents from cigarettes than someone who is passively exposed to cigarette smoke that may just be in the room, itself.

Q. But you're talking about how environmental tobacco smoke is much more dilute than mainstream cigarette smoke. Is environmental tobacco smoke also more dilute than sidestream tobacco smoke?

MR. BROOKEY: I'll object. That mischaracterizes the prior testimony. It's vague and ambiguous, but he can answer.

THE WITNESS: Well, if I understand your question, sidestream smoke would only -- could be considered as a surrogate for environmental tobacco

smoke because it doesn't contain exhaled mainstream smoke. What comes out when someone exhales after smoking -- after inhaling mainstream smoke are many of the similar constituents, but they may be in different ratios and certainly the thought is that potentially the number of -- the amount of nicotine may be -- may be lower than found in sidestream smoke; particulate number or particulate size range may be altered by inhaling smoke because some of it is going to be retained. Not all of it will be exhaled.

MR. KODSI: Q. Let me ask it this way:

In your experiments where you study tobacco smoke in animals, you use a surrogate that you referred to as aged and diluted sidestream smoke.

How does aged and diluted sidestream smoke differ from just pure sidestream smoke?

A. Well, I think it's probably more characteristic to what true environmental tobacco smoke is because, typically, environmental tobacco smoke is going to be those constituents of the smoke that are lingering in a room or in the air for a period of time, where there's going to be some changes that will occur, and there will be some coalescence potentially of particles, depending upon the concentration of the particulates, and so I think that aged and diluted sidestream smoke is more reminiscent of what truly environmental tobacco smoke is compared to just sidestream smoke that's not diluted and not aged.

15:30:41 1 Q. Could you define for me what you mean by
15:30:43 2 "aged"?
15:30:46 3 A. In our studies that are all for experimental
15:30:50 4 purposes, aging occurs in a chamber where the smoke,
15:30:56 5 the sidestream smoke that has been collected from the
15:30:59 6 smoldering cigarette, is drawn up into a dilution
15:31:04 7 chimney. Then it is introduced into a chamber where it
15:31:07 8 is actually mixed with fresh filtered air and --
15:31:12 9 through the use of a fan, and that aging process will
15:31:15 10 occur anywhere from two to four minutes before a
15:31:20 11 portion of that air is drawn off and further diluted
15:31:23 12 with fresh air before it's introduced into animal
15:31:28 13 chambers for our studies.
15:31:30 14 Q. And does sidestream smoke also age in real
15:31:36 15 world environments?
15:31:38 16 A. Yes.
15:31:41 17 Q. Now, in -- let's talk about your
15:31:44 18 declaration. You make some statements about ETS in
15:31:47 19 there. Exhibit 528, I believe.
15:31:51 20 Paragraph 12 on Page 4, in the first
15:32:01 21 sentence, you indicate that there -- over 3800
15:32:05 22 different constituents are found in ETS.
15:32:10 23 Have there been that many constituents
15:32:12 24 actually identified in environmental tobacco smoke, to
15:32:19 25 your knowledge?
15:32:19 26 A. My understanding is that there are probably
15:32:24 27 well over 3800 different constituents in tobacco smoke.
15:32:30 28 In fact, I think there has been the suggestion that

15:32:33 1 there may actually be a fivefold greater number than
15:32:40 2 actually are indicated here, but they represent perhaps
15:32:42 3 a small fraction of that.
15:32:43 4 So in terms of exactly the number of
15:32:49 5 constituents that have chemically been identified, I'm
15:32:53 6 not actually sure, but I'm under the assumption that a
15:33:00 7 fair number of these have been identified.
15:33:06 8 Q. You used the phrase "tobacco smoke" just
15:33:08 9 now.
15:33:08 10 A. Uh-huh.
15:33:10 11 Q. Do you know how many chemicals have been
15:33:11 12 identified in environmental tobacco smoke?
15:33:16 13 A. No.
15:33:22 14 Q. Is it possible that there are chemicals that
15:33:24 15 exist in mainstream tobacco smoke that do not exist in
15:33:28 16 environmental tobacco smoke?
15:33:30 17 MR. BROOKEY: Objection; calls for
15:33:33 18 speculation, but he can answer.
15:33:37 19 THE WITNESS: I don't really know the answer
15:33:38 20 to that. Although, as you probably know, the
15:33:44 21 generation of mainstream cigarette smoke occurs when
15:33:49 22 the burning temperature is very different compared to
15:33:52 23 sidestream smoke, and so due to that burning
15:33:58 24 characteristic of being very different, there may
15:34:01 25 potentially be some constituents that are uniquely
15:34:04 26 generated during that phase of the combustion of a
15:34:10 27 cigarette that may be different from environmental
15:34:13 28 tobacco smoke.

15:34:14 1 MR. KODSI: Q. Yes. I noticed in
15:34:15 2 Paragraph 11 of your declaration just above the point
15:34:18 3 we were talking about, you state that -- I highlighted

15:34:27 4 the line, but -- that environmental tobacco smoke has a
15:34:30 5 chemical composition that is strikingly different from
15:34:33 6 that found in mainstream smoke.
15:34:39 7 It's Lines 6 through 8.
15:34:42 8 A. Uh-huh.
15:34:43 9 Q. What did you mean by that?
15:34:46 10 A. My understanding of this is that the ratio
15:34:49 11 of constituents found within sidestream smoke compared
15:34:55 12 to mainstream smoke can be very different. Sometimes
15:34:59 13 they can be close to being identical, but often times
15:35:03 14 you see them in very different ratios. So if you
15:35:06 15 compare the constituent in sidestream smoke to
15:35:10 16 mainstream smoke, often times it will be in a higher
15:35:14 17 ratio compared to mainstream smoke. So that's what I'm
15:35:18 18 referring to when I made that statement.
15:35:21 19 Q. What is the significance of different
15:35:23 20 constituent ratios?
15:35:26 21 A. Well, potentially, if you have a higher
15:35:30 22 ratio of certain constituents, they may actually elicit
15:35:35 23 a very different health effect compared to active
15:35:41 24 smoking. It also brings about the potential for
15:35:46 25 certain constituents that may have -- that have known
15:35:51 26 health effects or a response on biological systems that
15:35:56 27 if it's going to be in a higher concentration that
15:35:59 28 effect may actually be more adverse than if you were

15:36:04 1 just exposed to mainstream cigarette smoke.
15:36:07 2 Q. And I want to make sure that the record is
15:36:11 3 clear, and I want to also make sure that you and I are
15:36:14 4 talking on the same page.
15:36:15 5 When we're talking about constituent ratios,
15:36:22 6 I understand you to mean that if in environmental
15:36:22 7 tobacco smoke the typical nicotine particle ratio is
15:36:27 8 10 to 1, that if it's 2 to 1 in mainstream smoke, that
15:36:34 9 would be a different ratio. Is that what you're
15:36:36 10 referring to?
15:36:37 11 A. That's -- that's correct.
15:36:39 12 Q. And that's a hypothetical. I'm not trying
15:36:41 13 to say those numbers are accurate.
15:36:42 14 A. Uh-huh. Sure.
15:36:43 15 Q. It's just to understand the concept.
15:36:45 16 So if we have two chemicals in environmental
15:36:47 17 tobacco smoke that have a 10 to 1 ratio and in one of
15:36:54 18 your studies, for example, those -- when you used
15:36:57 19 levels of one TSP, you found that those two chemicals
15:37:01 20 actually had a 100 to 1 ratio, how would that affect
15:37:05 21 your study results as I understand what you're talking
15:37:08 22 about?
15:37:08 23 MR. BROOKEY: Objection; lack of foundation,
15:37:09 24 incomplete hypothetical. You can answer.
15:37:13 25 THE WITNESS: Well, again, as I mentioned, I
15:37:17 26 think that, if you have a higher ratio of certain
15:37:21 27 constituents that have known biological effects than
15:37:25 28 you have in mainstream smoke, then the response that

15:37:30 1 you would get with exposure to sidestream smoke may
15:37:32 2 actually be more pronounced, more prominent, than with
15:37:38 3 mainstream smoke.
15:37:41 4 Perhaps a good example of this -- but I am
15:37:43 5 not saying that this is something that we've proven,
15:37:45 6 but if you look at the amount of ammonia that's

15:37:49 7 generated in sidestream smoke versus mainstream smoke,
15:37:53 8 it's about 70 times higher in sidestream smoke than it
15:37:57 9 is in mainstream smoke. If we assume that -- if --
15:38:01 10 basically, we don't have to assume. We know that
15:38:02 11 ammonia is an irritant to the airways. This would be
15:38:06 12 an example where the sidestream smoke has the potential
15:38:12 13 to be far more irritating to the lung airways than the
15:38:17 14 mainstream smoke can.
15:38:19 15 MR. KODSI: Q. Are you familiar with any
15:38:20 16 environmental tobacco smoke measurements of ammonia?
15:38:26 17 A. From the literature.
15:38:29 18 Q. You are familiar with them?
15:38:30 19 A. Yes.
15:38:31 20 Q. And you haven't seen any environmental
15:38:33 21 tobacco smoke measurements of ammonia that are higher
15:38:36 22 than mainstream smoke?
15:38:40 23 A. That's why I gave the example.
15:38:42 24 Q. I just wanted to make sure I understood.
15:38:43 25 A. Okay. Okay.
15:38:44 26 Q. You're talking about sidestream smoke
15:38:46 27 ammonia is more concentrated than mainstream --
15:38:48 28 A. That's correct.

15:38:49 1 Q. -- but when people have tried to measure
15:38:51 2 ammonia in environmental tobacco smoke, they get levels
15:38:54 3 that are lower than mainstream.
15:38:56 4 A. And, again, that would be based on the fact
15:38:58 5 that environmental tobacco smoke concentrations are
15:39:01 6 going to be lower than mainstream smoke concentrations.
15:39:07 7 Q. And as we're talking about tobacco smoke
15:39:09 8 chemistry and levels that have been measured in the
15:39:13 9 environment, is this an area that you would recognize
15:39:14 10 Dr. Roger Jenkins to have expertise in?
15:39:20 11 A. I think he is. Certainly, he has been the
15:39:24 12 person that when we set up our exposure system and when
15:39:28 13 we were trying to determine the sorts of things that we
15:39:30 14 should measure that he was the person who -- who gave
15:39:34 15 us the advice that we felt to be very reasonable, and
15:39:38 16 we followed many of his suggestions.
15:39:43 17 Q. Are you familiar with the book that he has
15:39:45 18 written on ETS chemistry and exposures?
15:39:47 19 A. Yes, I am.
15:39:49 20 Q. Have you reviewed that book?
15:39:51 21 A. Yes.
15:39:52 22 Q. Actually, have you seen the newest version
15:39:53 23 that just came out last month?
15:39:56 24 A. No.
15:39:57 25 Q. Okay.
15:39:57 26 A. No.
15:39:58 27 Q. He -- at the time that -- the book you're
15:40:00 28 talking about is the one that he wrote in 1992.

15:40:03 1 A. That's correct. Uh-huh.
15:40:05 2 Q. At that time, would you agree that that was
15:40:06 3 the most comprehensive -- had the most comprehensive
15:40:12 4 data available on ETS chemistry and exposures?
15:40:14 5 A. Uh-huh. I think, much like the EPA document
15:40:18 6 of 1992, that was a great source to go to if you wanted
15:40:23 7 to know about the chemistry of environmental tobacco
15:40:26 8 smoke.
15:40:26 9 Q. And you're -- when you say, "that," you're

15:40:28 10 talking about the book that Dr. Jenkins authored?
15:40:31 11 A. Right, with Mike Guerin.
15:40:33 12 Q. Right, with Mike Guerin and Bruce Thompkins.
15:40:37 13 A. Uh-huh.
15:40:37 14 Q. And you're aware also that that book was
15:40:39 15 done under CIAR funding?
15:40:41 16 A. Yes.
15:40:56 17 Q. Okay. Let's just talk a little bit about --
15:40:58 18 we've talked about ETS chemistry a little bit. I just
15:41:01 19 want to make sure we have the same definition of
15:41:04 20 "exposure." How do you define "exposure to ETS"?
15:41:13 21 A. Well, it's by inhalation.
15:41:17 22 Typically, again, though, we would need to
15:41:20 23 be talking about which -- if we're talking about
15:41:23 24 perinatal development that it's not always going to be
15:41:26 25 by inhalation for -- for the newborn, especially, you
15:41:34 26 know, during the gestational period.
15:41:38 27 Q. Let me make sure I understood that, and I
15:41:41 28 don't mean to interrupt you --

15:41:42 1 A. Uh-huh.
15:41:43 2 Q. -- but you said, "for the newborn."
15:41:44 3 Once the baby is born, how would they be
15:41:47 4 exposed to ETS by a route other than inhalation?
15:41:52 5 A. Well, they could also be exposed to
15:41:54 6 constituents of ETS through nursing, through the
15:42:00 7 mother's milk.
15:42:01 8 Q. Through maternal exposure to ETS?
15:42:06 9 A. Basically, if the mother is also being
15:42:08 10 exposed to environmental tobacco smoke, you'd have
15:42:14 11 that, but I guess what I meant in discussing this is
15:42:18 12 that, when you asked me how would exposures occur,
15:42:25 13 well, primarily it would be by direct inhalation of
15:42:26 14 environmental tobacco smoke, but during pregnancy, it's
15:42:31 15 the mother that's exposed, and then those -- then there
15:42:35 16 can be potential exposure to the fetus by way of the
15:42:39 17 placenta. So that's actually what I was referring to.
15:42:44 18 Q. How does exposure differ from concentration?
15:42:54 19 A. Well, again, if I understand your question
15:42:58 20 correctly, "concentration" really is just a measure of
15:43:08 21 how much is in the air based on some constituent or
15:43:14 22 some property that you're characterizing the exposure
15:43:19 23 by, but "exposure," itself, is basically whatever is in
15:43:26 24 the air. That's what you'll breathe.
15:43:30 25 So I guess I don't find a real connection
15:43:32 26 between "exposure" and "concentration."
15:43:34 27 Q. I've heard that exposure -- and let's just
15:43:36 28 see if you agree with this -- that exposure is equal to

15:43:39 1 concentration times duration, that you need to know
15:43:43 2 what you're exposed to; you need to know the
15:43:46 3 concentration plus the amount of time you're in that
15:43:48 4 environment.
15:43:49 5 A. Uh-huh.
15:43:49 6 Q. Is that a fair characterization of how
15:43:52 7 exposure may differ from just looking at concentration
15:43:54 8 alone?
15:43:55 9 A. Yes. That's accepted as an exposure
15:44:01 10 parameter.
15:44:03 11 Q. How does "exposure" differ from "dose"?
15:44:12 12 A. Well, again, to me, I guess the dose would

15:44:15 13 be basically what is -- what you have been exposed to
15:44:23 14 over a period of time. So I'm wondering if "dose" is
15:44:31 15 somewhat like -- I'm sorry -- the concentration times
15:44:35 16 time phenomenon that you're referring to.
15:44:38 17 Q. Would dose also incorporate an analysis of
15:44:41 18 the amount that actually makes it into the body and
15:44:43 19 into a particular target organ?
15:44:46 20 A. If we're talking about dose to specific
15:44:50 21 organ systems, yes.
15:44:54 22 Q. And, in your animal studies, have you
15:44:56 23 calculated exposure or dose?
15:45:01 24 A. We've really calculated exposure.
15:45:05 25 Q. But you haven't looked at the dose of any
15:45:08 26 tobacco smoke constituent that makes it to any of the
15:45:11 27 target organs?
15:45:13 28 A. No, not as something that is measured.

15:45:21 1 Q. Could you describe for me the factors that
15:45:22 2 might affect ETS exposures in human environments?
15:45:31 3 A. Well, certainly the -- whether you're in a
15:45:39 4 location where someone is smoking, how many cigarettes
15:45:45 5 that individual may be smoking, how much time the
15:45:50 6 individual may spend in a room where other people are
15:45:54 7 smoking.
15:45:56 8 Q. The degree of ventilation in that
15:45:58 9 environment?
15:45:59 10 A. That's correct.
15:45:59 11 Q. The individual person's respiratory rate?
15:46:03 12 A. That's correct.
15:46:03 13 Q. The size of the environment?
15:46:05 14 A. That's correct, and also I think it's
15:46:08 15 important, you know, that goes along with the
15:46:11 16 ventilatory rate is also the metabolic function of that
15:46:18 17 individual and also the fact that children are going to
15:46:24 18 be different from adults in terms of just exactly what
15:46:30 19 exposure they receive based on their age, on their --
15:46:37 20 the stage of their development also.
15:46:40 21 Q. You say that children metabolize
15:46:44 22 environmental tobacco smoke differently from adults?
15:46:47 23 A. They metabolize many things differently from
15:46:49 24 adults.
15:46:50 25 Q. And actually metabolizing environmental
15:46:55 26 tobacco smoke would be different even between different
15:46:57 27 children?
15:46:59 28 A. There can be that kind of variation from one

15:47:02 1 person to the next, yes.
15:47:03 2 Q. Because there are several individual
15:47:05 3 characteristics that dictate how we metabolize all
15:47:09 4 agents, including ETS?
15:47:11 5 A. Uh-huh. That's correct.
15:47:16 6 Q. Let's shift to talking about some principles
15:47:18 7 of toxicology, if you could describe for me generally
15:47:25 8 in your view how animal experiments are used to study
15:47:29 9 health effects.
15:47:34 10 A. Well, I think that animal studies are
15:47:36 11 essential because of the fact that they allow us to
15:47:40 12 very precisely monitor the conditions of exposure to
15:47:46 13 actually generate conditions that we can characterize
15:47:51 14 very well and that we can control how long those
15:47:55 15 exposures may be or the timing for the exposures.

15:48:02 16 They also provide us with the ability to
15:48:07 17 look at different parameters that we wouldn't be able
15:48:10 18 to do in human studies. It allows us not only to do
15:48:17 19 measures at just looking at functional parameters, but
15:48:24 20 it also allows us to look at the degree of growth, the
15:48:29 21 degree of cellular maturation, differentiation. It
15:48:36 22 allows us to look at whether there are changes in cell
15:48:39 23 proliferation during the period of exposure to
15:48:44 24 environmental tobacco smoke and whether those are
15:48:46 25 different based on the age of the animal or the
15:48:50 26 concentration of the environmental tobacco smoke that
15:48:55 27 we're generating.
28 (Ms. Moore leaves the room.)

15:48:58 1 MR. KODSI: Q. Now, you mentioned in there,
15:49:00 2 I think, that you thought animal experiments were
15:49:02 3 important because they provided -- and these may be my
15:49:05 4 words not yours -- but a controlled environment?
15:49:08 5 A. That's correct.
15:49:10 6 Q. Because, in animal experiments, you can
15:49:13 7 control conditions that you can't otherwise control in
15:49:15 8 human environments?
15:49:19 9 A. That's right. It's just simply a fact that
15:49:23 10 you're always going to find different degrees,
15:49:27 11 different concentrations of environmental tobacco smoke
15:49:32 12 in society, and those are going to be produced by all
15:49:38 13 sorts of different brands of tobacco products that
15:49:46 14 cannot really be considered to be a standard or a
15:49:49 15 reference for environmental tobacco smoke.
15:49:52 16 Q. And by having a controlled environment in
15:49:59 17 animal studies, it helps you to eliminate what is
15:49:59 18 referred to in epidemiology as "confounding factors"?
15:50:04 19 A. That's correct.
15:50:05 20 Q. And you recognize that in the human studies,
15:50:07 21 the epidemiology studies, there are several confounding
15:50:10 22 factors that affect those studies?
15:50:13 23 A. That's correct.
15:50:13 24 Q. And in the epidemiology studies that you've
15:50:18 25 talked about earlier with respect to ETS, you're aware
15:50:21 26 that they don't actually measure environmental tobacco
15:50:25 27 smoke exposures?
15:50:28 28 A. Do you mean personal --

15:50:29 1 Q. Yes.
15:50:30 2 A. -- exposures? That's correct.
15:50:33 3 Q. What is your understanding of how the
15:50:34 4 epidemiology studies of ETS actually measure exposures?
15:50:42 5 A. Well, certainly a personal history is taken,
15:50:44 6 a questionnaire, asking about a smoking history in the
15:50:51 7 home or in the workplace, if for children, you know,
15:50:56 8 whether it's the mother or the father that is the
15:51:01 9 smoker and, again, how much smoking occurs.
15:51:10 10 Certainly some studies actually include
15:51:12 11 measurements, field measurements, for the levels of
15:51:17 12 environmental tobacco smoke based on typically
15:51:20 13 particulate concentrations in the home.
15:51:23 14 Q. Are you aware of epidemiology studies on the
15:51:26 15 effects of ETS on children that actually took airborne
15:51:34 16 measurements of ETS constituents for the children being
15:51:37 17 studied in that study?
15:51:38 18 A. For each child?

15:51:39 19 Q. Yes.
15:51:40 20 A. No, I'm not aware.
21 (Ms. Moore rejoins the proceedings.)
15:51:43 22 MR. KODSI: Q. Are you aware of some that
15:51:44 23 did it for some children? I mean how -- when you say,
15:51:49 24 "each child" --
15:51:50 25 A. Uh-huh.
15:51:52 26 Q. -- why don't you describe for me the
15:51:53 27 epidemiology studies you're aware of that actually did
15:51:55 28 some sort of field measurement.

15:51:58 1 A. Well, actually, these are typically for
15:52:03 2 looking at children who may have had studies where
15:52:09 3 they've -- that maybe there's been an admission to the
15:52:13 4 hospital for a respiratory infection or there's been
15:52:18 5 some history of respiratory problems and potential for,
15:52:25 6 you know, wheezing or a cough or some sort of
15:52:30 7 bronchitis, and a few of those studies have potentially
15:52:37 8 done some monitoring of levels of particulate matter or
15:52:44 9 ETS in the home, but I think they're very limited.
15:52:49 10 In fact, I guess the other approach that
15:52:51 11 one -- that has been taken has been to measure
15:52:57 12 biomarkers for exposure, such as measuring cotinine or
15:53:03 13 nicotine in the urine or the saliva or plasma of
15:53:11 14 individuals as a way of trying to assess better
15:53:14 15 exposure to environmental tobacco smoke, but I think
15:53:17 16 those kind of measures really are very limited in
15:53:22 17 really giving you a very clear picture of what the
15:53:25 18 actual exposure history is or exposure profile is.
15:53:31 19 Q. And you would agree that studies that use
15:53:33 20 questionnaire data to assess ETS exposure are not as
15:53:37 21 accurate in assessing exposure as if they'd actually
15:53:40 22 measured it?
15:53:42 23 MR. BROOKEY: Objection; incomplete
15:53:43 24 hypothetical, lacks foundation, calls for speculation.
15:53:45 25 He can answer.
15:53:49 26 THE WITNESS: I think there's some
15:53:50 27 limitations with -- with questionnaires.
15:53:56 28 I think that there is going to be a certain

15:54:02 1 degree of error that's present in those, but based on
15:54:08 2 what is available, given some of the limitations for
15:54:14 3 epidemiological studies, it's probably one of the most
15:54:19 4 reasonable and logical approaches to take with the
15:54:22 5 assumption that you may have some degree of error, but
15:54:28 6 it's your best estimate of exposure assessment.
15:54:32 7 MR. KODSI: Q. Are you familiar with the
15:54:32 8 concept of "recall bias" with respect to exposure
15:54:35 9 assessment in epidemiology studies?
15:54:39 10 A. No.
15:54:44 11 Q. Okay.
15:54:44 12 A. Although can I ask it? Does that mean
15:54:46 13 basically people recalling what happened that morning
15:54:50 14 or that -- or last week or --
15:54:55 15 Q. Right. It's a -- let me ask you:
15:54:57 16 Are you familiar with a concept that
15:54:59 17 addresses people's ability to recall their exposures
15:55:02 18 using questionnaires and the biases that pertain to
15:55:07 19 that ability? Is that a concept you're familiar with?
15:55:10 20 A. No.
15:55:18 21 Q. Now, we've talked about the advantage of

15:55:21 22 animal studies in that they provide a controlled
15:55:24 23 environment. When conducting animal studies, I think
15:55:27 24 we talked about using a particular substance to study
15:55:31 25 with the animal. Is that called the "test material"?
15:55:35 26 A. Yes.
15:55:35 27 Q. Is that -- that's one thing we call it?
15:55:37 28 A. Yeah. Yeah. It would be.

15:55:40 1 Q. And is the test material you select an
15:55:42 2 important part of a study protocol?
15:55:46 3 A. Yes.
15:55:47 4 Q. For example, if you want to study the
15:55:49 5 effects of benzene, you would use benzene as your test
15:55:53 6 material?
15:55:54 7 A. Yes.
15:55:56 8 Q. Is this analysis we just went through any
15:55:58 9 different when we're talking about studying complex
15:56:00 10 mixtures?
15:56:04 11 A. Well, I think that environmental tobacco
15:56:08 12 smoke certainly could be classified as a "complex
15:56:12 13 mixture," and due to that fact, I think it's very
15:56:19 14 important to be able to control as closely as possible
15:56:25 15 how you generate something like environmental tobacco
15:56:33 16 smoke, and of course for our studies, that's going to
15:56:36 17 have to consist of the surrogate of sidestream
15:56:40 18 cigarette smoke that's been aged and diluted.
15:56:44 19 So from the perspective of using the same
15:56:48 20 type of cigarette, of burning the cigarettes -- well,
15:56:55 21 actually, even one step previous, of conditioning the
15:57:06 22 cigarettes prior to smoking them and then smoking them
15:57:06 23 in a very precise manner and using a system that allows
15:57:10 24 for diluting and aging of the smoke in exactly the same
15:57:16 25 manner is important if you're going to be able to do
15:57:19 26 anything more than once and try to say that there is
15:57:23 27 some association.
15:57:25 28 Q. And the points that you're just making go to

15:57:27 1 the importance of reproducibility of data, correct?
15:57:30 2 A. That's correct. Uh-huh.
15:57:31 3 Q. That you want to follow standard protocols
15:57:34 4 so that, if someone else tries to do the same
15:57:36 5 experiment, they'll be able to reproduce your results?
15:57:40 6 A. That's correct.
15:57:40 7 Q. And the protocols you were just describing
15:57:41 8 are protocols that deal with the cigarette preparation
15:57:44 9 prior to even using cigarettes in exposure studies?
15:57:47 10 A. That's correct.
15:57:47 11 Q. And some of those protocols actually have
15:57:50 12 been developed through the FTC method, correct?
15:57:53 13 A. That's correct.
15:57:55 14 Q. Now, we were talking about studying complex
15:57:58 15 mixtures, and I think you were saying that you want to
15:58:02 16 study something as similar to ETS as possible.
15:58:06 17 A. That's correct.
15:58:07 18 Q. In your opinion, is it ever appropriate just
15:58:09 19 to look at one constituent when trying to study the
15:58:12 20 effects of a complex mixture such as ETS?
15:58:17 21 MR. BROOKEY: Objection; incomplete
15:58:19 22 hypothetical, lacks foundation. He can answer.
15:58:21 23 THE WITNESS: I would answer "yes" if you're
15:58:23 24 especially interested in trying to determine the role

15:58:27 25 that that constituent may play in a health effect. It
15:58:33 26 also becomes more important -- it becomes important if
15:58:38 27 you're trying to define a mechanism for that effect.
15:58:45 28 MR. KODSI: Q. Is it possible that you may

15:58:47 1 see effects from one constituent alone that will not
15:58:52 2 occur when that constituent is placed in the complex
15:58:56 3 mixture?
15:58:57 4 MR. BROOKEY: Same objections.
15:59:03 5 THE WITNESS: There's a possibility that
15:59:04 6 that could possibly occur.
15:59:06 7 MR. KODSI: Q. Are you familiar with
15:59:07 8 studies that have looked at the effects of -- well, let
15:59:11 9 me just back up for a second.
15:59:12 10 Are you familiar with the tobacco specific,
15:59:14 11 and I trust them to be known as "NNK"?
15:59:18 12 A. Yes.
15:59:18 13 Q. Are you familiar with studies that have
15:59:19 14 looked at the effects of NNK alone compared with the
15:59:22 15 effects of NNK plus tobacco smoke?
15:59:26 16 A. No.
15:59:27 17 Q. That's not a body of literature you're
15:59:32 18 familiar with?
19 A. (Shakes head.)
15:59:34 20 MR. KODSI: Okay. Let's mark this one.
21 (Whereupon, Defendants' Exhibit 533 was
22 marked for identification.)
15:59:59 23 MR. KODSI: Q. Doctor, I've handed you what
16:00:00 24 has been marked as Exhibit 533. Do you recognize this?
16:00:04 25 A. Yes.
16:00:05 26 Q. Okay. And this is a peer-reviewed paper
16:00:09 27 titled "Sidestream Cigarette Smoke Generation and
16:00:12 28 Exposure System From Environmental Tobacco Smoke

16:00:16 1 Studies," correct?
16:00:18 2 A. Yes.
16:00:18 3 Q. And this is a paper on which you were one of
16:00:21 4 the authors?
16:00:22 5 A. That's correct.
16:00:22 6 Q. And Dr. Roger Jenkins is also an author on
16:00:27 7 this paper, correct?
16:00:28 8 A. Yes.
16:00:28 9 Q. And this is a paper that was done
16:00:29 10 pursuant -- done under CIAR funding?
16:00:47 11 A. Yes.
16:00:48 12 Q. Let me see the last page.
16:00:50 13 A. (Complies.)
16:01:11 14 MR. KODSI: Let me go off the record for
16:01:13 15 just one second.
16:01:14 16 THE VIDEOGRAPHER: Going off the record, the
16:01:15 17 time is 4:01.
16:01:19 18 (Recess taken)
16:16:21 19 (Ms. Moore is not present.)
16:16:53 20 THE VIDEOGRAPHER: Back on the record, the
16:16:54 21 time is 4:16.
16:16:57 22 MR. KODSI: Q. Dr. Pinkerton, you've got
16:16:59 23 Exhibit 533 in front of you, and I think we just
16:17:02 24 finished talking about what that was.
16:17:05 25 Could you just generally describe for me
16:17:07 26 what the purpose of this paper was?
16:17:10 27 A. We felt it was important since we had no

16:17:13 28 previous publication record with studies with

16:17:17 1 environmental tobacco smoke to simply publish in a
16:17:22 2 stand-alone article that would be peer-reviewed, the
16:17:25 3 approach that we took for generating sidestream
16:17:28 4 cigarette smoke for environmental tobacco smoke
16:17:31 5 studies.
16:17:33 6 Q. And so was the purpose of this paper to
16:17:35 7 explain the approach you took to generate aged and
16:17:39 8 diluted sidestream smoke in your animal studies?
16:17:43 9 A. That's correct. We wanted to show that
16:17:47 10 these studies could be done without a tremendous amount
16:17:52 11 of expense and that just simply by having the proper
16:17:56 12 resources and the equipment that you could generate
16:18:03 13 aged and diluted sidestream smoke as a surrogate for
16:18:07 14 environmental tobacco smoke.
16:18:11 15 Q. How did you go about developing this chamber
16:18:14 16 and smoke development method?
16:18:17 17 A. This was under the guidance of Dr. Jenkins
16:18:23 18 who had previous experience with doing experimental
16:18:28 19 generation of environmental tobacco smoke before, so we
16:18:34 20 had him as a subcontract investigator on our CIAR grant
16:18:44 21 and asked him to come and visit us after the workshop
16:18:49 22 that we had back in Maryland, sponsored by CIAR, and to
16:18:56 23 look at our system of exposure chambers and just
16:19:03 24 exactly the sorts of things that we would need to -- to
16:19:06 25 generate environmental tobacco smoke.
16:19:10 26 So he was instrumental in helping us to
16:19:13 27 acquire an Arthur D. Little 2 smoking machine and also
16:19:20 28 in building a dilution chimney for us, and together we

16:19:26 1 sat down and talked about what would work out best and
16:19:29 2 actually came to an agreement that we needed to build a
16:19:33 3 double system to make sure that we wouldn't have
16:19:37 4 variation based on the fact that we were smoking a
16:19:41 5 single cigarette at a time. So we actually created a
16:19:44 6 double smoking system and dilution chimneys so that we
16:19:53 7 could stagger our cigarettes, and we could smoke two at
16:19:56 8 a time.
16:19:59 9 Roger, again, gave us quite a bit of
16:20:06 10 direction on the sorts of things that we should be
16:20:06 11 measuring, such as total particulate concentration,
16:20:09 12 carbon monoxide levels and nicotine levels, so he
16:20:13 13 helped us to establish the methodology that we used to
16:20:19 14 determine nicotine from air samples that we collected
16:20:22 15 from the chambers using special collecting filters.
16:20:30 16 He also agreed to do some extended analyses
16:20:35 17 of the different constituents of the cigarette smoke
16:20:40 18 that we were not capable of doing ourselves, but we
16:20:44 19 felt it would be essential just to simply show what was
16:20:48 20 our exposure chamber, what kind of concentrations were
16:20:52 21 we generating and the exposure chamber, both in the
16:20:56 22 conditioning chamber as well as in the exposure
16:20:58 23 chamber, and where they fell in terms of concentrations
16:21:03 24 measured in field studies, so...
25 (Ms. Moore rejoins the proceedings.)
16:21:06 26 MR. KODSI: Q. Now, you mentioned that you
16:21:09 27 measured Total Suspended Particulates, carbon monoxide
16:21:14 28 and nicotine.

16:21:15 1 A. Routinely.
16:21:16 2 Q. Yes. What was the purpose behind picking
16:21:18 3 those three constituents to measure?
16:21:20 4 A. In discussing all of the various things that
16:21:22 5 we could measure, it was felt that these three
16:21:26 6 constituents would allow us to best characterize and
16:21:31 7 demonstrate consistency in our exposures on a daily
16:21:36 8 basis.
16:21:36 9 The Total Suspended Particulate matter, we
16:21:40 10 knew that was -- only could have been generated by the
16:21:43 11 sidestream smoke that we were collecting, aging and
16:21:46 12 diluting.
16:21:46 13 The same was true for the carbon monoxide as
16:21:51 14 well as for the nicotine. We knew that the primary
16:21:54 15 source for that nicotine would be from combusting the
16:22:00 16 cigarettes and collecting the smoke. Although, we did
16:22:05 17 find that exposure chambers do allow for nicotine
16:22:09 18 plating out onto surfaces which then can later lead to
16:22:14 19 some off-gassing of nicotine from chamber surfaces.
16:22:21 20 Q. I was going to ask you about that.
16:22:23 21 If you could turn to Page 92, I think you
16:22:25 22 discuss there the phenomenon that you just described
16:22:28 23 about nicotine claiming out.
16:22:31 24 A. Uh-huh.
16:22:32 25 Q. In the first full paragraph on Page 92 of
16:22:34 26 Exhibit 533, it starts with it was during -- if you
16:22:39 27 could just kind of read that to yourself and I was
16:22:41 28 going to ask you to explain what you're discussing in

16:22:44 1 that paragraph.
16:22:53 2 A. When we first started doing these studies,
16:22:55 3 we found that as we measured the nicotine that there
16:23:00 4 appeared to be a trend for an increase in concentration
16:23:05 5 of the nicotine that we would collect from the exposure
16:23:08 6 chambers, and this was rather perplexing to us until we
16:23:14 7 discussed this further among ourselves as well as with
16:23:18 8 Dr. Jenkins, and he pointed out the fact that because
16:23:22 9 of a limited surface that -- surface area that we might
16:23:28 10 actually -- plating out of nicotine is a common event
16:23:32 11 that is going to -- it's just that kind of substance
16:23:34 12 that will collect onto any surfaces that it comes into
16:23:37 13 contact and will stick, but because our exposure
16:23:42 14 chambers were of a limited surface area that we were
16:23:46 15 probably seeing a point of saturation with nicotine
16:23:51 16 plating out onto the surfaces that then would allow for
16:23:55 17 subsequent off-gassing of the nicotine into the
16:23:59 18 chambers, and so that could possibly be part of the
16:24:05 19 reason why we saw kind of a general increase in our
16:24:08 20 nicotine concentrations over time.
16:24:10 21 We were also a bit surprised that our
16:24:13 22 nicotine concentrations seemed to be within the range
16:24:17 23 but always somewhat on the high side of what field
16:24:21 24 studies would say. If you have a given particulate
16:24:25 25 concentration of environmental tobacco smoke, then your
16:24:31 26 nicotine particulate ratio should be this, and we
16:24:35 27 tended to always be on the high side, and so that was
16:24:39 28 part of our reasoning for why we thought that may be

16:24:44 1 occurring.
16:24:47 2 So in order to alleviate that problem, we
16:24:50 3 went through a process of cleaning the chambers, the

16:24:54 4 inner surfaces of the chambers, weekly to -- in an
16:24:59 5 attempt to remove as much of accumulated smoke
16:25:04 6 constituents as we could, including the nicotine,
16:25:08 7 itself.
16:25:08 8 Q. Now, you indicated that the nicotine
16:25:12 9 particulate ratios in your test chamber were higher
16:25:15 10 than what you'd expect to see in real world field
16:25:18 11 studies, and you were talking about how to alleviate
16:25:22 12 that problem. Why did you view that as a problem?
16:25:24 13 A. Well, again, our objective was to try to
16:25:27 14 create as close to the same type of exposure
16:25:35 15 composition that would exist in a room that a person
16:25:41 16 potentially could be exposed to environmental tobacco
16:25:45 17 smoke in. So we didn't really consider it to be a
16:25:47 18 problem other than just the fact that we wanted to try
16:25:51 19 to reproduce as closely as we possibly could what was
16:25:56 20 really happening if a person was smoking in a room, and
16:26:01 21 we even discussed, you know, different things that
16:26:05 22 could be done and immediately discarded them, such as
16:26:10 23 carpeting the chambers and doing things like that,
16:26:13 24 hanging drapes and things like that, that might help to
16:26:16 25 bring about that adjustment in the nicotine
16:26:21 26 concentrations, but obviously, we didn't go that far.
16:26:29 27 Q. And that would also provide, I guess, a more
16:26:29 28 comfortable atmosphere for the rats as well, right?

16:26:30 1 A. That's right. They would feel more at home.
16:26:32 2 Q. And you talked about you wanted to get as
16:26:35 3 close to real world exposure composition.
16:26:38 4 What do you mean by "exposure composition"?
16:26:42 5 A. What I mean is what happens in the home in
16:26:48 6 terms of if there's someone in the home who is smoking,
16:26:53 7 the type of environmental tobacco smoke that one would
16:26:56 8 be exposed to there. That's what we really wanted to
16:27:00 9 try to produce in our animal studies.
16:27:03 10 Q. And if the constituent ratios in your
16:27:07 11 chamber were substantially different from the
16:27:09 12 constituent ratios in real world environments, then you
16:27:13 13 might not be studying ETS?
16:27:17 14 A. Well, I'm not sure I would take it that
16:27:21 15 far --
16:27:23 16 Q. Okay.
16:27:23 17 A. -- because in looking at the various
16:27:25 18 constituents that were measured that went beyond just
16:27:27 19 the three major ones, we found that what was in the
16:27:32 20 exposure chambers, the ones we did -- went through the
16:27:36 21 dilution process, is that the majority of those were
16:27:41 22 right within the range of what has been measured in
16:27:44 23 field studies.
16:27:45 24 Q. What other constituents did you measure?
16:27:48 25 A. They were primarily particulate phase
16:27:53 26 constituents, and those are found, I believe, in a
16:27:56 27 table, Table 1 on Page 86.
16:28:02 28 So these were selective vapor phase

16:28:05 1 constituents, and we measured these both in the
16:28:10 2 conditioning chamber -- this is where the smoke is aged
16:28:13 3 and diluted -- as well as in the exposure chamber,
16:28:16 4 which housed the animals for -- during the exposure
16:28:22 5 periods.
16:28:24 6 Q. And one of the reasons that you have Table 1

16:28:26 7 in that paper is to address the issue you and I were
16:28:29 8 just talking about -- correct? -- to see that the
16:28:32 9 ratios among these constituents in your chamber were
16:28:36 10 similar to the ratios that you would find in real world
16:28:40 11 field studies?
16:28:41 12 A. Yeah. I wouldn't call them "ratios."
16:28:44 13 I would call them the "range of
16:28:45 14 concentrations" that -- because in terms of the ETS
16:28:50 15 measurements in the field studies, those are actual
16:28:53 16 measurements expressed in micrograms per cubic meter
16:28:58 17 rather than a ratio of one constituent compared to
16:29:01 18 another or relative to how much is found in
16:29:05 19 environmental tobacco -- or in sidestream smoke versus
16:29:09 20 mainstream smoke.
16:29:11 21 Q. Right. And, when I'm talking about ratios,
16:29:13 22 I'm not talking about sidestream versus mainstream.
16:29:16 23 A. Uh-huh.
16:29:16 24 Q. Let's look at this chart, Table 1, for
16:29:20 25 example, that in your chamber, you would expect the
16:29:24 26 ratio between acrylonitrile and benzene to be similar
16:29:30 27 to the acrylonitrile-benzene ratio found in real world
16:29:36 28 environments?

16:29:39 1 A. That's what would be expected, but if you
16:29:42 2 look at what's in real world environments for those two
16:29:46 3 things, you can see that they're -- well, for the -- is
16:29:52 4 it the acetonitrile that you were pointing out?
16:29:54 5 Q. Acrylonitrile.
6 A. Okay.
16:29:56 7 Q. We could use either one.
16:29:58 8 A. Okay. Well, basically you can see that
16:29:59 9 there is anywhere from more than from a ten- to
16:30:06 10 twenty-fold difference in the range, and so I think
16:30:09 11 that -- that it's difficult to know exactly what those
16:30:16 12 ratios would be for two selected different constituents
16:30:20 13 to say, oh, well, this one should always be higher or
16:30:23 14 lower, but...
16:30:26 15 Q. What is the highest concentration that you
16:30:30 16 verified that these constituents remained at similar
16:30:34 17 concentrations?
16:30:37 18 A. The highest concentration would be with the
16:30:43 19 particulate concentration being at 4 milligrams. That
16:30:46 20 was in the conditioning chamber.
16:30:47 21 Q. Did you conduct -- have you conducted a
16:30:53 22 verification for these constituents at a concentration
16:30:56 23 higher than 4 milligrams?
16:30:58 24 A. I have not.
16:31:00 25 Q. Are you aware -- now, you are aware of the
16:31:05 26 exposure studies done by Dr. Witschi?
16:31:09 27 A. That's correct.
16:31:10 28 Q. Are you aware if he has done such a

16:31:12 1 verification when he has used concentrations as high as
16:31:15 2 a 130 milligrams per cubic meter?
16:31:20 3 A. Yes, he has, I think as high as maybe
16:31:22 4 90 milligrams that he has done, and those are reported
16:31:25 5 in the literature. I may be a coauthor on one of those
16:31:29 6 papers.
16:31:29 7 Q. For all of these constituents?
16:31:32 8 A. No. Those were measured by a laboratory on
16:31:42 9 the campus at University of California, Davis and not

16:31:47 10 with Dr. Jenkins' help. So they, perhaps, selected
16:31:51 11 other constituents other than these. There may be some
16:31:54 12 overlap.
16:31:56 13 Q. Are you aware of any of the papers that
16:31:58 14 Dr. Witschi has published where he talks about
16:32:01 15 concentrations for constituents other than CO, TSP and
16:32:07 16 nicotine?
16:32:11 17 A. Well, there's -- there's the one study in
16:32:13 18 which he -- in which he removed the particulate phase
16:32:20 19 from the sidestream cigarette smoke, and then he looked
16:32:25 20 at the unfiltered sidestream smoke and measured
16:32:30 21 constituents there, that were beyond those three that
16:32:32 22 you mentioned, as well as measuring the same
16:32:35 23 constituents when he had removed the particulate phase
16:32:40 24 from that.
16:32:41 25 Q. In those papers, he measured constituents to
16:32:43 26 demonstrate how low they were and that they really no
16:32:46 27 longer existed in his exposure chamber, right?
16:32:48 28 A. That was the purpose for that --

16:32:50 1 Q. Right.
16:32:51 2 A. -- analysis.
16:32:51 3 Q. But he hasn't measured any of these
16:32:56 4 constituents in Table 1 to show the concentration that
16:32:59 5 his animals are exposed to in his -- in the studies
16:33:11 6 where he hasn't removed the vapor or particulate
16:33:11 7 phases?
16:33:11 8 A. That's right.
16:33:11 9 Q. Okay.
16:33:11 10 A. That's not a consistent measurement that he
16:33:13 11 does.
16:33:22 12 Q. Okay. And I think you addressed this same
16:33:24 13 point in Paragraph 16 of your declaration that we've
16:33:27 14 been talking about, Page 6.
16:33:45 15 A. That's correct.
16:33:45 16 Q. That's where you addressed the nicotine
16:33:47 17 fluctuation issue?
16:33:49 18 A. That's right, and we also further elaborated
16:33:53 19 on the fact that the humidity and temperature may also
16:34:02 20 affect the amount of nicotine that one detects in air
16:34:06 21 samples taken from the chambers.
16:34:09 22 Q. Now, we've been -- this whole time we've
16:34:11 23 been talking about principles of toxicology. We
16:34:13 24 started by talking about the test material and talking
16:34:18 25 about how animal studies are used.
16:34:22 26 The next thing I'd like to talk about is
16:34:24 27 route of administration. How important is the chosen
16:34:27 28 route of administration in an animal study?

16:34:35 1 A. Well, I think that that is important.
16:34:42 2 Again, with our studies with environmental
16:34:45 3 tobacco smoke, we really have to say that the route of
16:34:51 4 administration dependent upon the timing for
16:34:54 5 development is different. We typically -- as one who's
16:35:04 6 interested in environmental air pollutants, I typically
16:35:07 7 think the major route of administration of an air
16:35:11 8 pollutant that I want to understand its health effects
16:35:15 9 would be directly by inhalation of the substance.
16:35:19 10 With environmental tobacco smoke, we have
16:35:23 11 sufficient studies to show that the route of
16:35:29 12 administration is not just confined to -- by inhalation

16:35:35 13 but that there is something about maternal exposures
16:35:41 14 which are by inhalation of the mother, that there is
16:35:45 15 passage of some constituents -- whatever those are we
16:35:50 16 don't know -- but that clearly have an impact on the
16:35:53 17 fetal lung development that -- and then in combination
16:35:57 18 with further exposure postnatally by the direct
16:36:03 19 inhalation of environmental tobacco smoke of those --
16:36:06 20 those newborns leads to changes that we measure that
16:36:13 21 are significant and that we don't see if the exposures
16:36:20 22 are only by inhalation postnatally.
16:36:25 23 Q. And when you talk about the exposure of the
16:36:27 24 mother, although the unborn child isn't exposed by
16:36:31 25 inhalation --
16:36:32 26 A. Uh-huh.
16:36:33 27 Q. -- when you study the animals and expose the
16:36:35 28 mother rat via inhalation, you're trying to mimic human

16:36:40 1 exposure scenarios?
16:36:41 2 A. That's correct, passive exposures.
16:36:45 3 Q. And that was a point I just wanted to see if
16:36:47 4 I'm understanding correctly, if you'll turn to Page 4
16:36:50 5 of your declaration, Paragraph 13 at the bottom.
16:36:56 6 You indicate that an important consideration
16:36:58 7 for any experimental study is to produce conditions
16:37:01 8 which are similar to those that individuals may be
16:37:04 9 exposed.
16:37:05 10 Is that the point that we're talking about
16:37:06 11 here is that you want to try to mimic human exposure
16:37:09 12 conditions?
16:37:10 13 A. That's correct.
16:37:11 14 Q. Both to the appropriate test material and
16:37:16 15 the route of administration?
16:37:18 16 A. That's correct.
16:37:19 17 Q. And also I would assume that the
16:37:21 18 concentration should try to mimic human exposure
16:37:24 19 conditions as much as possible?
16:37:27 20 A. That would be the ideal of any experiment.
16:37:33 21 Q. Now, are you familiar with the term "no
16:37:35 22 observable effect level"?
16:37:37 23 A. Yes.
16:37:37 24 Q. And we'll -- again, for abbreviation
16:37:40 25 purposes, we'll refer to that as a "NOEL" from now on,
16:37:45 26 N-O-E-L.
16:37:46 27 A. Uh-huh.
16:37:46 28 Q. What is an "N-O-E-L"?

16:37:48 1 MR. BROOKEY: I object to the extent it
16:37:48 2 calls for a legal conclusion, but he can answer.
16:37:50 3 THE WITNESS: Okay. "NOEL" is basically
16:37:57 4 exposure to some sort of constituent where no
16:38:01 5 observable effect is detected.
16:38:08 6 MR. KODSI: Q. And "NOEL" is a scientific
16:38:13 7 term of art, correct?
16:38:14 8 A. That's correct.
16:38:15 9 Q. And so although it may also be a legal term,
16:38:18 10 when you and I talk about it today, we'll be talking
16:38:20 11 about it in the scientific context; is that fair?
16:38:23 12 A. Okay.
16:38:23 13 Q. Okay. So the definition that you just gave
16:38:25 14 me would be the scientific definition, as you
16:38:28 15 understand it, of "NOEL"?

16:38:29 16 A. Yes.
16:38:31 17 Q. Okay. What does -- what do NOELs tell you
16:38:33 18 about exposures to different chemicals?
16:38:39 19 A. Well, my impression and interpretation would
16:38:42 20 be that if you do have a NOEL observed that you have a
16:38:50 21 threshold, at which constituents that may lead to some
16:38:57 22 sort of health effect at higher concentrations, if they
16:39:02 23 are sufficiently low, do not give -- do not manifest
16:39:07 24 those health effects.
16:39:17 25 Q. And what does that tell you when you're
16:39:18 26 trying to extrapolate from animal exposures to human
16:39:22 27 exposures?
16:39:27 28 A. Well, my understanding would be that a NOEL

16:39:29 1 level would actually allow one to start to formulate a
16:39:36 2 safety margin or a buffer at which there may be some
16:39:43 3 degree of protection; although it can never be absolute
16:39:46 4 that it's going to protect everyone.
16:39:50 5 Q. But -- although, as I understand it,
16:39:52 6 scientists don't like to speak in terms of absolutes.
16:39:55 7 The existence of an exposure level at which you don't
16:39:58 8 find an observable effect at least supports the concept
16:40:03 9 of a threshold?
16:40:07 10 A. That's correct.
16:40:07 11 Q. And it supports the concept of a threshold
16:40:09 12 at or around the level at which you find no effect?
16:40:15 13 A. That's correct.
16:40:19 14 Q. Now, I want to talk a little bit about
16:40:22 15 animal models, and what I mean by "animal models" is
16:40:25 16 the decision of which animal to use in an animal
16:40:35 17 experiment. What is the decision-making process you go
16:40:35 18 through in deciding which animal to use in a given type
16:40:36 19 of experiment?
16:40:36 20 A. Well, I think it's important to do a
16:40:39 21 literature review for the type of animals that have
16:40:45 22 been used in the past for such studies.
16:40:50 23 One also has to look at the questions that
16:40:53 24 one wishes to address. If this is something where
16:40:59 25 there are time constraints or resources, limited
16:41:06 26 resources, one has to take into consideration what
16:41:09 27 would be the best animal model to accommodate those --
16:41:15 28 those budget restrictions.

16:41:19 1 I think also it's important to keep in mind
16:41:23 2 that one know the differences between the animal model
16:41:26 3 and if there are chemical or structural or functional
16:41:31 4 differences and -- between that animal model and the
16:41:37 5 human as a way of better being able to decide how much
16:41:43 6 or what can be extrapolated from those animal studies
16:41:47 7 to human studies.
16:41:50 8 Also I think it's important to keep in mind
16:41:53 9 does this animal model that one selects -- does it show
16:41:59 10 similarities in its response to whatever constituent
16:42:06 11 that you're going to be studying. Would you see those
16:42:08 12 same effects in the human population?
16:42:13 13 Q. Now, in most of your -- not all because we
16:42:16 14 talked about this earlier, but in most of your studies,
16:42:18 15 you have used rats --
16:42:20 16 A. That's correct.
16:42:21 17 Q. -- as your animal model.
16:42:22 18 What is the decision-making process in

choosing rats over mice over guinea pigs?
A. Well, there are a number of reasons.
One is that the literature is really very complete in terms of lung development in using rats as a model for understanding the multi-step process of maturation and differentiation and morphogenesis of the lungs.
It's been very well characterized in terms of its timing, just the gestational period is very consistent and very short, a period of 21-and-a-half

days for gestation.
Also postnatal development is extremely well established in the rat with most alveolarization or development of new air sacs in lungs finished within the first three to four weeks of life, which allows us to have a very reasonable amount of time or a window of exposure that we can use as a way of trying to look at effects during critical windows of development that would also be the same types of development that we would see in the human respiratory system.
Q. Now, you mentioned you're doing a recent study in which you're looking at Rhesus monkeys.
A. That's correct.
Q. What was the decision-making process you underwent in transitioning from rats to Rhesus monkeys?
A. The reason for that is because we have only human epidemiological studies suggesting or showing health effects with environmental tobacco smoke, and then we have our animal model as the rat, and there is nothing in between.
So we felt that if studies in nonhuman primates would be something that would be important to establish -- we know what happens in humans; we know what happens in rats in quite good detail because we can look at cellular changes, functional changes, and we felt that we needed to look at another species to see if -- where it fell in the spectrum of between rat to human in terms of responses to environmental tobacco

smoke.
Q. And the Rhesus monkey studies are being conducted as we speak, correct?
A. That's correct.
Q. And you feel those are necessary in order to further understand the role that ETS may play in these disease end points?
A. Well, I'm not relying on those studies for -- because of the fact that those are not published. I think they will help further our understanding of the effects of environmental tobacco smoke on perinatal development of the lungs.
MR. KODSI: Let me mark...
(Whereupon, Defendants' Exhibit 534 was marked for identification.)
MR. KODSI: Q. Dr. Pinkerton, I've handed you what has been marked as Exhibit 534, which is an abstract that you published with Dr. Joad in the American Review of Respiratory Disease in 1992; is that correct?
A. That's correct.

16:46:43 22 Q. And that abstract on Page A91 is titled
16:46:46 23 "Environmental Tobacco Smoke Effects on Pulmonary
16:46:51 24 Function and Airway Reactivity in Developing Rats."
16:46:53 25 A. Yes.
16:46:54 26 Q. And I wanted to just ask you to help me
16:46:56 27 clarify something.
16:46:57 28 At the end of that abstract, the last

16:47:00 1 sentence right before you indicate the funding sources,
16:47:04 2 you indicate, "We conclude that chronic exposure to ETS
16:47:09 3 does not appear to alter lung function, airway
16:47:13 4 reactivity or lung or body weights in developing rats
16:47:16 5 unless the rat is not a good species to use as a model
16:47:19 6 for studying ETS effects on pulmonary function and
16:47:23 7 airway reactivity in children."
16:47:29 8 A. This was a study that we did. It's one of
16:47:31 9 our first, initial studies to look at the effects of
16:47:36 10 environmental tobacco smoke in the postnatal lung.
16:47:41 11 These studies were actually initiated with exposures
16:47:51 12 beginning within the first or second day of life after
16:47:51 13 the birth of the rat pups and continued on until they
16:47:56 14 were -- I think we did studies both at 7 weeks and
16:48:00 15 then -- I'm sorry. Yeah -- 7 weeks or 50 days of age
16:48:04 16 and then again around 100 to 120 days of age, and in
16:48:09 17 these particular studies, we found no effects of
16:48:16 18 exposure to environmental tobacco smoke on any of these
16:48:20 19 parameters.
16:48:23 20 Subsequent to these studies, we designed
16:48:28 21 some to begin exposures with the pregnant dam and then
16:48:34 22 continued those exposures after the birth of the
16:48:37 23 animals postnatally to 100 days. Actually, these were
16:48:44 24 to approximately 8 weeks of age, around 70 days,
16:48:51 25 something like that, and in those studies, we actually
16:48:55 26 compared critical windows of exposure where we again
16:48:58 27 compared only postnatal exposure or only in utero
16:49:03 28 exposure or the combination of the two.

16:49:07 1 And it was from those studies that we found
16:49:10 2 that these rats responded with -- that had pre and
16:49:15 3 postnatal exposure to environmental tobacco smoke, that
16:49:18 4 they showed significant airway reactivity. I think the
16:49:23 5 statement that we made obviously was premature in
16:49:32 6 saying that the rat was not a good model. Our exposure
16:49:36 7 conditions were not a good model for what actually
16:49:41 8 happens with children with exposure to environmental
16:49:46 9 tobacco smoke or at least for a number of children,
16:49:50 10 so...
16:49:52 11 Q. And you would agree that -- maybe then that
16:49:54 12 this conclusion provides an example that science and
16:49:59 13 conclusions in science develop over time?
16:50:02 14 A. That's correct.
16:50:04 15 Q. That sometimes, as scientists, you make a
16:50:07 16 conclusion that you later learn is incorrect?
16:50:09 17 A. That's correct.
16:50:12 18 Q. Now, in concluding that the rat was not a
16:50:16 19 good model for studying ETS effects on pulmonary
16:50:22 20 function and airway reactivity in children, once you
16:50:25 21 reached that conclusion in 1992, why did you continue
16:50:27 22 to use the rat model?
16:50:30 23 A. Actually, because there were many other
16:50:33 24 effects that we were actually seeing that were --

showed that the rat was a very good model, and I think that because we felt it was important as we looked at, you know, how are children actually exposed to cigarette smoke, what are the things that happen, that

we really began to realize that it was more than just direct exposure of the child after its birth, that exposures were also potentially occurring during pregnancy, either if the mother was a smoker herself or being passively exposed to cigarette smoke.

So I think, in evaluating where we wanted to go with these studies -- because we also did studies in guinea pigs, especially under Dr. Joad's direction -- that we did have other animal species that we were considering for these studies, but when it came to really looking at perinatal lung development and the potential effects of an exposure to environmental tobacco smoke, the rat still represented the best animal species for us to do those studies. We could also have extended those to the mouse, but pulmonary function testing in those at this time would have been very difficult in such a small animal as that.

Q. Okay. Now, would you agree that the Rhesus monkey would be a better test animal for studying health effects in humans than would the rat?

MR. BROOKEY: Objection; incomplete hypothetical, lacks foundation, compound, but he can answer.

THE WITNESS: I think that if we could do -- well, the answer -- simple answer is, yes, because the monkey -- in terms of its anatomical composition and makeup of the respiratory system as well as its gestational period. Its postnatal development is much

more closely aligned to human development than the rat is.

MR. KODSI: Q. And I guess I shouldn't have limited my question to just the Rhesus monkey.

You would agree that that would be your opinion about all nonhuman primates that you could conduct studies with?

MR. BROOKEY: Same objections. He can answer.

THE WITNESS: Uh-huh. Well, again, I would agree with that.

I think that what is important, though, is that all our answers are not going to be found in working with nonhuman primates. I think that there are many things that we can understand, and we would be -- we would do a better job at understanding them because I think we would be more responsible in terms of, you know, respecting the importance of, you know, trying to -- well, we had very big reservations about using monkeys for our studies. It took us seven years to come to the conclusion that we didn't really have any other alternative but to use another species and especially the monkeys, but even though we've now started research with Rhesus monkeys, our primary research will continue in the rodent model, particularly with rats.

MR. KODSI: To shortly change topics and

16:54:24 28 then we'll call it a day, what I'd like to do is we

16:54:27 1 need to mark -- whatever the best way to mark this
16:54:29 2 stack is. Actually, to put a sticker on it is the best
16:54:34 3 way, but we'll identify that as one giant exhibit, and
16:54:40 4 I don't... you can go ahead and put a sticker on it,
16:54:43 5 and I'll shut up, but we can go off the record for a
16:55:08 6 second.
16:55:22 7 THE VIDEOGRAPHER: Going off the record, the
16:55:22 8 time is 4:55.
16:55:23 9 (Discussion held off the record)
10 (Whereupon, Defendants' Exhibit 535 was
11 marked for identification.)
16:56:00 12 THE VIDEOGRAPHER: Back on the record, the
16:56:01 13 time is 4:56.
16:56:04 14 MR. KODSI: Q. Doctor, we have a stack of
16:56:05 15 materials here that has been marked as Exhibit 535, and
16:56:09 16 I want to ask you if you recognize what is there. I
16:56:14 17 know there's a lot of material, but if you can give me
16:56:16 18 a general description if that looks familiar to you,
16:56:18 19 then we can kind of walk through it.
16:56:27 20 A. Yes, it does. It contains my declaration,
16:56:34 21 my contract agreement with CIAR, my Curriculum Vitae,
16:56:50 22 the declaration statement of Dr. Slotkin, and also my
16:56:59 23 agreement for consulting services.
16:57:03 24 Would you like me to just --
16:57:05 25 Q. No. I was going to stop you there. Rather
16:57:07 26 than identify -- because we'll walk through that, I
16:57:10 27 think, tomorrow, some of those individually, but if you
16:57:13 28 could generally -- if you want to just flip through it

16:57:14 1 yourself real quick to see it's what you remember your
16:57:18 2 file being... did he do this or did you all do this?
16:57:25 3 MR. CAFFERTY: I got that from John McGuire
16:57:27 4 by fax on Friday. It looks like it got a little closed
16:57:31 5 up. I think four and five are jammed together.
16:57:39 6 THE WITNESS: Just looking through the top
16:57:42 7 of this, these are all of my files that I possibly have
16:57:44 8 on this case.
16:57:47 9 MR. KODSI: Q. Okay. And did you provide
16:57:48 10 those files pursuant to a request from one of the
16:57:51 11 lawyers in this case?
16:57:52 12 A. Yes.
16:58:13 13 Q. Okay. Whatever the next number is --
16:58:33 14 A. Okay. Yeah.
16:58:34 15 Q. -- that looks like your file for this case?
16:58:37 16 A. Yes, including a lot of my publications,
16:58:40 17 so...
16:58:46 18 Q. All right. Is there anything that you have
16:58:47 19 in your file related to this case that is not in
16:58:49 20 Exhibit 535, to your knowledge?
16:58:53 21 A. Not to my knowledge.
22 (Whereupon, Defendants' Exhibit 536 was
23 marked for identification.)
16:58:54 24 MR. KODSI: Q. Okay. And let me just show
16:58:55 25 you really quickly what has been marked as Exhibit 536
16:58:58 26 and ask you if you've seen that document before.
16:59:01 27 A. Yes, I have.
16:59:02 28 Q. Now, that is the Deposition Notice for you

for this case, correct?
A. Yes.
Q. If you'd turn to the last page, there is an Exhibit A which requests that you provide certain documents. Is that something you've seen before?
A. Yes.
Q. Was this generated in response to the requests in Exhibit A?
A. Yes.
MR. BROOKEY: Actually, yes. I just need to say it's a little bit vague and ambiguous because the expert witness file that you were indicating was generated during the course of his expert work.
MR. KODSI: Good point, Brian.
MR. BROOKEY: Do you mean Exhibit 536 in the form that it was delivered to you?
MR. KODSI: Right. That's a very good point.
MR. BROOKEY: Okay.
MR. KODSI: I agree. Yes.
Q. Is Exhibit 535 provided to me or provided to your attorneys in response to Exhibit A?
A. Yes.
Q. Okay. Could you walk me through your selection process of what to include in Exhibit 535? and I'll make that easier for you. If the answer is that you just put together your entire case file and threw -- and didn't leave anything out, that's all I'm

asking.
A. And that's the answer.
Q. Okay.
A. Uh-huh.
Q. Was there any e-mail or computer correspondence that you chose not to put in Exhibit 535 that might be relevant to this case?
A. No.
Q. Okay. And what you're telling me is, as we read through -- and we don't need to go through this out loud -- all the documents that are described in Exhibit A -- and if you want to read it to yourself, and why don't you, if you could just read Exhibit A to yourself and tell me if there are any documents that would be responsive to those five requests that are not in Exhibit 535, and that should be the last question I have on this.
A. And that's correct. I think this contains all of the things that are listed under Exhibit A.
MR. KODSI: Okay. Well, it's 5:00 o'clock. I just wanted to get that one on the record. I think today's a good stopping point, and I'll shoot for my best to get done as early as I can. I'm thinking, if we start at 8:30, we'll shoot for maybe an hour or two after lunch, but we won't keep you here until 5:00, I don't think, and beat the heavy Sacramento traffic.
THE VIDEOGRAPHER: This marks the end of Tape Number 3 in the deposition of Kent Pinkerton.
Going off the record, the time is 5:01.
(Whereupon, the deposition was recessed at 5:01 p.m., to be continued on Tuesday, May 23, 2000, at the hour of 8:30 a.m.)
I declare under penalty of perjury that the

foregoing is true and correct. Subscribed at _____, California, this _____ day of _____, 2000.

_____
Kent E. Pinkerton

CERTIFICATE OF REPORTER

I, SHANNON TAYLOR-SCOTT, a Certified Shorthand Reporter, hereby certify that the witness in the foregoing deposition was by me duly sworn to tell the truth, the whole truth and nothing but the truth in the within-entitled cause;

That said deposition was taken down in shorthand by me, a disinterested person, at the time and place therein stated and that the testimony of the said witness was thereafter reduced to typewriting, by computer, under my direction and supervision;

I further certify that I am not of counsel or attorney for either or any of the parties to the said deposition, nor in any way interested in the event of this cause, and that I am not related to any of the parties thereto.

DATED: June 5, 2000

_____
SHANNON TAYLOR-SCOTT, RPR, CSR 10067